IN THE SUPREME COURT OF MISSISSIPPI

JEFFREY KEITH HAVARD, *Petitioner*

v. **No. 2013-DR-01995-SCT**

STATE OF MISSISSIPPI, *Respondent*

RESPONDENT'S EXHIBITS

VOLUME 1: EXHIBITS A – F

VOLUME 2: EXHIBITS G– H

VOLUME 3: EXHIBITS H1– L

VOLUME 4: EXHIBITS M-O

VOLUME 5: EXHIBIT Q

IN THE CIRCUIT COURT OF ADAMS COUNTY, MISSISSIPPI

STATE OF MISSISSIPPI	RECEIVED AND FILED SEP 18 2002 M.L. VINES, CIRCUIT CLERK BY _____ D.C.	PLAINTIFF
VERSUS		C.# 02-KR-0141-J
JEFFERY HAVARD		DEFENDANT

MOTION FOR PRODUCTION OF MEDICAL RECORDS

COMES NOW Jeffery Havard, by and through his attorneys of record, and moves this Court to order the State of Mississippi, or any medical providers, to produce all medical records of the deceased child, Chloe Madison Britt, D/O/B 8/29/01, and in support hereof would respectfully show, to-wit:

1. The alleged cause of death of Chloe Madison Britt was "shaken baby syndrome" according to Dr. Stephen Hayne, the State's medical expert.
2. Upon information and belief, there are other medical problems and/or conditions that could cause a deceased infant to appear to have died from "shaken baby syndrome," such as medication and/or vaccinations.
3. Defendant needs said infants' medical records in order to prepare a proper defense to his capital murder charge herein.

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EXHIBIT tabbies A

IN THE CIRCUIT COURT OF ADAMS COUNTY, MISSISSIPPI

STATE OF MISSISSIPPI

VS.

JEFFREY KEITH HAVARD

RECEIVED
AND FILED
SEP 25 2002
M.L. VINES, CIRCUIT CLERK
BY_____ D.C.

NO. 02-KR-0141 - J

DEFENDANT

ORDER

This cause came before the court this date on motion of the defense for independent evaluation of autopsy report. The defense requests the court to appoint a qualified medical expert to review and evaluate the autopsy report prepared by Dr. Steven T. Hayne, pathologist. The court finds that the report of Dr. Hayne, and any supplements, are in the possession of the defendant, and that Dr. Hayne is available to answer any questions that defense counsel may have of him. The court finds no basis presented to it to order a medical expert to conduct an independent evaluation of the autopsy report.

SO ORDERED, this the 25th day of September, 2002.

Forrest A. Johnson

FORREST A. JOHNSON
CIRCUIT JUDGE

(Order Prepared by Court)

EXHIBIT
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B

351 Wis.2d 682
Unpublished Disposition
See Rules of Appellate Procedure, Rule 809.23(3), regarding citation of unpublished opinions. Unpublished opinions issued before July 1, 2009, are of no precedential value and may not be cited except in limited instances. Unpublished opinions issued on or after July 1, 2009 may be cited for persuasive value.
NOTE: THIS OPINION WILL NOT APPEAR IN A PRINTED VOLUME.
THE DISPOSITION WILL APPEAR IN A REPORTER TABLE.
Court of Appeals of Wisconsin.

STATE of Wisconsin, Plaintiff–Respondent,
v.
Michael L. CRAMER, Defendant–Appellant.

No. 2012AP2547–CR. | Oct. 15, 2013.

Appeal from a judgment and an order of the circuit court for Milwaukee County: Kevin E. Martens and Jeffrey A. Wagner, Judges. *Affirmed.*

Before FINE, KESSLER and BRENNAN, JJ.

Opinion

¶ 1 FINE, J.

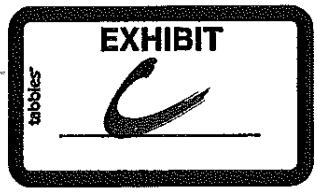
*1 Michael L. Cramer appeals the judgment entered on a jury verdict convicting him of first-degree reckless homicide, *see* WIS. STAT. § 940.02(1), and the circuit court's order denying his motion for postconviction relief. [1] Cramer contends: (1) the State "presented demonstrably false and misleading testimony at the trial that violated" his right to due process; (2) he should get a *Machner* hearing on his claim that his trial lawyer gave him constitutionally deficient representation; and (3) he is entitled to a new trial in the interest of justice. *See State v. Machner,* 92 Wis.2d 797, 285 N.W.2d 905 (Ct.App.1979) (hearing to determine whether lawyer gave a defendant ineffective assistance). We affirm.

I.

¶ 2 In February of 2009, the State charged Cramer with physical abuse of a child because his ten-week-old son, Matthew, who had been in Cramer's care, came to the hospital with "acute bleeding around the brain, subdural hemorrhaging, retina bleeding behind both eyes, linear bruising to the left arm and left thigh area and consistent abusive head trauma." When Matthew arrived at Children's Hospital, he had a pulse, but needed help breathing and was in "a comatose state."

¶ 3 Cramer told Milwaukee police detective, Ronald Taylor, that he had been caring for Matthew and his three-year-old daughter, Camariana, because Cramer's wife, Candace, left at 8:30 a.m. to run errands. Cramer said Matthew seemed "perfectly healthy." Cramer said he fed Matthew at 11:30 a.m., but could not burp him. Cramer told the officer that he then put Matthew face down on the couch so he could take a shower. Cramer said that Matthew "appeared agitated" and "was fidgeting and moving around." Cramer said that he found Matthew limp and nonresponsive about fifteen to twenty minutes later. Cramer told the officer that he then started cardiopulmonary resuscitation and called 911.

¶ 4 Medical help arrived and, after twenty to thirty minutes, got Matthew's "pulse back." Paramedic Stephanie Hampton removed Matthew's diaper "to get some idea of how long this baby ha[d] been" "[n]ot breathing, no pulse, baby dead," and found "the



stool in the diaper was cold." Hampton also noticed "bruising there on [Matthew's lower] leg" "[a]nd ... some other bruising, upper body bruising[.]" The paramedics took Matthew to Children's Hospital.

*2 ¶ 5 Mrs. Cramer told the police that Matthew had been acting normally the night before this incident, explaining that she fed, burped, and changed Matthew at 3:45 a.m. and 6:55 a.m. She said that Matthew was awake when she left the house that morning and that she did not see any bruises on him. When police confronted Cramer about the medical evidence showing that Matthew "had sustained brain injuries that were consistent with blunt force trauma," Cramer told police "that he didn't know right from wrong anymore and didn't know himself anymore"; that "he wanted to tell what happened, but that he wanted to tell his wife first but that he didn't know how to tell her." When the officers told Cramer that he would have to tell them "the entire truth" before he could meet with his wife, Cramer refused, saying "he would be judged no matter what he said that he would be viewed as a child abuser."

¶ 6 Matthew died in September of 2009 after the hospital removed him from life support. Milwaukee County medical examiner, Dr. Wieslawa Tlomak concluded that Matthew died from "complications of blunt force injuries of the head[,]" and ruled the death a homicide. The State amended the charge against Cramer to first-degree reckless homicide. At the trial, the State called Dr. Thomas Valvano, who treated Matthew as an "attending child abuse physician[ ] at Children's Hospital." Dr. Valvano testified, as material:

- He does evaluations on children who come to the hospital with "injuries [that] we don't know how they happened or we are worried that they may have been inflicted injuries."
- "Matthew was a two[-]month old who had a severe brain injury without any clear explanation as to why that happened[.]"
- Mrs. Cramer told him that Matthew had not been in any car accidents or had fallen, and that Matthew had not been sick but was "an active, healthy, normal acting baby."
- He examined Matthew and found that he was non-responsive and had fixed and dilated pupils. He also testified that "Matthew had a linear bruise to his left arm and also had bruises above and below his left knee, and those were important because he's only two months old. So he's not walking and running and playing like a toddler, and so he's not doing anything that should result in a bruise. Unless something is done to him, he shouldn't have any bruise.... So they were a sign that Matthew had sustained some trauma."
- Matthew's bruises suggested that the child "sustained some intentional injury," in light of no "history of any accidental injury."

*3 • "[T]he CT scan showed that Matthew had bilateral, meaning on both sides of his brain, subdural hemorrhages. So that means that there was bleeding in the subdural space over the front part of his brain on both sides."

- "[B]y the time we got the MRI done" "you could still see the subdural hemorrhages" and "we could now see significant swelling of the brain that had evolved from this initial brain injury that Matthew had sustained." "[W]e also saw small petechial hemorrhages within the brain tissue itself[,]" and "there was injury to the brain stem itself."
- "[W]hen we see trauma to the head, we often see in association with that head trauma bleeding in the retina." "Matthew had extensive retinal hemorrhages in both eyes" in "a very specific pattern that has very few causes."
- "[W]here the neck ends and the upper back starts, so at that part of the spine and the spinal cord there was a hematoma, essentially a swelling and collection of blood ... a bruise to the spine."
- Based on this, Dr. Valvano told the jury that his "opinion was that Matthew had sustained abusive head trauma." "These injuries take significant force. This isn't trauma from normal handling of a child. This isn't trauma from an accidental injury. This isn't symptoms of an infection or a bleeding disorder or suffocation, accidental suffocation, or all of the

other things that we considered that didn't fit. This taken as a whole all of these injuries indicated that Matthew had been abused." (Formatting altered.)

- Dr. Valvano testified that he ruled out sudden infant death syndrome as a cause because "you don't see bruising. You don't see retinal hemorrhages. You don't see these subdural hemorrhages and petechial hemorrhages to the brain tissue itself. [Sudden infant deaths] look very, very different than Matthew looked."

*4 • Dr. Valvano also told the jury that he did not "think that the injuries I saw are the result of the [e]ffects of having resuscitated him and having him brought back to life." "We see [the injuries that Matthew had] in children whose head has sustained these sort of rotational acceleration, deceleration kind of forces to the brain as a result of trauma."

- Dr. Valvano also told the jury that he could not "tell you the exact mechanism of what was physically done to him[,]" but "[t]hese injuries are the result of what we call rotational acceleration deceleration forces to the brain" "either because someone throws the baby down or throws the baby across the room or bangs the baby's head against something or hits the baby's head against a sofa cushion or shakes the baby or a combination of those things, the head moves in this arc back and forth."

- He testified that "[w]e have a lot of experience with" "accidental falls or [kids who] get accidentally dropped" in hospitals "and it's been well studied." "These kids don't sustain brain injury from those short falls. They may have a bump on the head. They may have a little bit of focal bleeding. They may sometimes rarely have a skull fracture or a collar bone fracture or a bump or a bruise. But that kind of focal injury from one impact gives you a very sort of focal specific located injury, not a diffuse injury and not multiple injuries like Matthew had."

¶ 7 On cross-examination, Dr. Valvano testified:

- There was no evidence of external injury on the skull, no bruises on the skull or neck.

- "Oftentimes children with abusive head trauma present with absolutely no signs of external trauma, and that's not uncommon ... because you can have impact against, for example, a cushion like a mattress or a sofa cushion or a chair cushion, and that won't leave any external signs of injury necessarily. But that's still a force from sudden deceleration that gets transmitted to the brain and that injures the brain even though it leaves no external sign of injury."

- "Shaking could have been part of what happened to him, but it's only one of the different types of mechanisms I've described that causes these injuries." "Shaken baby syndrome is a subset of the types of trauma that can cause these injuries. So abusive head trauma includes shaken baby syndrome, but it also includes banging the child's head against a table or a wall or throwing a child against the room."

*5 • Shaken baby syndrome is "still used quite frequently; but as I said, it's a subset of abusive head trauma which is also a term that is being used to describe this because it more fully describes what may have happened to the child."

¶ 8 When asked about the "controversy in the medical field about shaken baby syndrome" Dr. Valvano responded:

> There really is no controversy outside the courtroom. The American Academy of Pediatrics, pediatricians, neurosurgeons, it's well accepted that violently shaking a baby causes injury to that baby. And outside of a few limited numbers of physicians, most of whom appear as defense witnesses, there's really no controversy about it.

¶ 9 When the defense lawyer asked Dr. Valvano about "biomechanical research on the amount of force it takes to injure the brain," Dr. Valvano told the jury:

There has been biomechanical modeling. It's very crude. It's very, very hard to recreate the complexities of the human brain and human neck in a doll model.

So, for example, one of the first studies ever done was a plastic doll head stuffed with wet cotton attached to a metal hinge. That hardly replicates a human baby.

So, yes, there is biomechanical modeling that have tried to estimate forces. And that work is ongoing, and what we find is as those models become more sophisticated the amount of force that we are seeing that is required to cause these injuries is actually less.

....

But nonetheless there is a substantial amount of research, and it's well accepted in the medical community that abusive head trauma is a very real thing.

¶ 10 There was then the following exchange between the defense lawyer and Dr. Valvano:

Q. Before you said that there wasn't really any research. Now you're saying there is but you just don't agree with it?

A. I'm confused. I never said there wasn't research supporting abusive head trauma.

Q. But there has been research about how you cause brain injury either forcible events or how much force it takes to cause brain injury; is that correct?

A. There are biomechanical modeling studies and also computer modeling studies that are trying to do that, yes. But those are fairly crude still.

Q. And you don't believe in them?

A. It's not that I don't believe in them. I think we are still learning from them; and as the models become more sophisticated, our information is better.

But you can't look at a doll head stuffed with cotton and say that the information you get from that is directly transferable to a human baby who has been subjected to abusive head trauma. Because the human baby is obviously much more complex than a plastic head stuffed with wet cotton.

*6 ¶ 11 Dr. Tlomak, the medical examiner, also testified for the State, as material:

- That she did the autopsy of Matthew and concluded the "cause of death was complications of blunt force injuries of the head."
- She "saw bilateral ... subdural hemorrhages that were overlying frontal, parietal, and lateral lobes of the brain[.]" "[S]ubarachnoid hemorrhage, diffuse retinal hemorrhages" that "are markers for significant brain injury."
- That she did not believe that Matthew died from sudden infant death syndrome "[b]ecause I had findings of remote subdural hemorrhage, remote subarachnoid hemorrhage, findings of severe brain injury, findings of remote retinal hemorrhages; and it's not consistent with SIDS. With SIDS death that autopsy findings are negative."
- She also told the jury that Matthew's death "was a homicide" because there was no "history of accidental type injuries;" and "[t]he amount of force required to cause this type of injuries is very large" like that caused "during high speed motor car accidents or falling from high buildings." "There were multiple studies done that showed falling from at least third floor, at least third floor can cause this type of injuries." "[F]alling from the short distance two, three, four feet will not cause a severe brain injury."

¶ 12 During his cross-examination of Dr. Tlomak, Cramer's trial lawyer criticized her for:

- relying on "no history of accidental injury" and that "[i]f someone had given you a history of accidental injury, than you could well have determined that was an accident?";
- for basing "a large part [of] your determination is really your opinion based on what other people told you?"; and
- for "not go[ing] out and investigat[ing] it yourself" and for "relying on what those other people tell you to be the truth?"

¶ 13 The defense lawyer then asked Dr. Tlomak if she "rule[d] out shaken baby syndrome?" Dr. Tlomak answered: "I don't use the term 'shaken baby syndrome' " because "[i]t's a very controversial term." She explained that: "The baby can die from being shaken, but it's—the cause of death is still blunt force injuries of the head.... Because when the baby is shaken, the baby's head will move back and forth, and it's not in the straight line. The head will go on both sides. At the same time, the brain is moving inside the head; and it causes this severe injuries in the brain, and that's why the babies will die."

*7 ¶ 14 Cramer's lawyer got Dr. Tlomak to admit that she did not "know how much force" it would take to cause Matthew's injuries, and charged in his cross-examination that Dr. Tlomak had "no idea at all." The lawyer also got Dr. Tlomak to say she "wasn't there" and she "cannot tell what happened to the child." Dr. Tlomak also testified in response to the defense lawyer's questions that she looks for fractures in head trauma cases, and "there was no evidence of fractures" here.

¶ 15 Dr. Thomas Young, a "self-employed ... forensic pathologist," testified as an expert witness for the defense. Dr. Young theorized that Matthew's injuries were the result of "resuscitated Sudden Infant Death Syndrome" or "complications of hypoxic ischemic encephalopathy due to an apparent life threatening event." He testified that when the heart and breathing stops and "if somebody happens to get there early enough and then start doing CPR trying to resuscitate the child, they may be able to get the heart functioning again." He explained that in those rare cases:

- "[T]here's usually been very, very severe brain damage by that point" because when "blood flow to the brain stops for a period of time" "tissue death starts" and when "the heart starts up again and there's blood flow that is resumed" "the blood vessels will get leaky" "and then you'll start to get some oozing of blood" that can result in subdural hemorrhaging.
- "And at that point, you still start to get collections of blood in the subdural space that are not under any kind of pressure. It's just that they are basically oozing, and they start to collect in the subdural space." And then the leaky blood "will basically shift with gravity" "along the spinal cord[.]"
- That a subdural hemorrhage "is not always due to trauma."
- Dr. Valvano and Dr. Tlomak "committed an error in terms of their determination of cause and manner of death."

¶ 16 Cramer's lawyer then asked Dr. Young a series of questions about short falls:

Q. [Dr. Tlomak] testified earlier that assume that she said that, for example, that a fall on a bathtub where you hit your head may not be fatal. Do you agree with that?

A. I disagree with that.

Q. Why is that?

A. Because people and children have had accidents there in home situations in which there have been falls, frequently unguarded falls. And they've died as a result of this. These are items that are well documented.

*8 Q. And in your experience as a pathologist, forensic pathologist, have you had these sorts of cases?

A. Yes.

¶ 17 Cramer's lawyer also asked about the "lack of evidence of trauma in this case" and Dr. Young answered: "What both of these doctors do is they reason backwards where they start with the evidence and they don't listen to any witnesses and they don't pay attention to any accounts. They just basically say, this child has a subdural hematoma, this child has retinal hemorrhages, therefore, it's child abuse." (Formatting altered.) According to Dr. Young, subdural hematoma could be caused by "an inborn error of metabolism" problems with "platelet functions or blood clotting" "hypoxic ischemic encephalopathy" and "[w]ide varieties of trauma mostly from impact." Dr. Young also offered a variety of other causes for "retinal hematoma" besides "child abuse."

¶ 18 When asked about "shaken baby syndrome" Dr. Young said "Shaken baby syndrome was basically proven false back in 1987." Dr. Young testified about his experience with blunt force trauma cases:

> In situations where I've seen subdural hemorrhages from trauma from impact, either in the form of somebody either being hit with a blunt object or basically falling, there's evidence of trauma.
>
> You see the deep bleeding in the scalp. You can see skull fractures. It takes quite a bit of force. It takes quite a bit of energy to cause a subdural hemorrhage from trauma.
>
> When you see that sort of thing, traumatic cases are traumatic looking. There's a problem basically when you are attributing something to trauma, and there's no evidence of trauma.

Dr. Tlomak testified during the State's rebuttal that the damages seen on Matthew's autopsy "were completely different" from an autopsy she did on a resuscitated sudden infant death case.

¶ 19 As we have seen, the jury found Cramer guilty. His postconviction motion claimed that "the state presented demonstrably false testimony to the jury, that trial lawyer was ineffective in failing to challenge the false testimony and that because of the false testimony the real controversy has not been fully tried." Cramer's motion relies on the post-trial opinions of forensic pathologist, Dr. John Plunkett, who, according to Cramer's motion, would testify that:

*9 • "[L]ucid intervals" of up to three days, which can occur with head trauma, make it impossible to determine when Matthew's head injury occurred.

- Dr. Valvano and Dr. Tlomak's conclusions were incorrect; "Matthew had no evidence of shaking or impact injury." "There is no experimental evidence that shaking can cause brain damage in an infant" only neck damage.
- "Dr. Valvano's conclusion that Matthew's pattern of retinal hemorrhages can only be due to abusive head trauma or an severe accidental trauma is speculation and is contradicted by research and case-report literature. There are no experimental studies that support this mechanism. In contrast, there are several experimental studies indicating that an increase in intracranial pressure is the cause for hemorrhage in these situations."
- Dr. Valvano's testimony on "low-level fall[s]" "is incorrect."
- "Dr. Valvano's claim that other than a few defense witnesses there is no controversy about SBS syndrome is wrong as demonstrated by several recent court cases and journal articles."
- "Dr. Valvano's dismissal of biomedical modeling as crude ... is incorrect."
- "Dr. Valvano was incorrect when he said" he could not put a number on the force necessary to cause Matthew's injury because "[t]here have been Federal Standards for *infant* head injury thresholds since approximately 1995."
- Dr. Tlomak's testimony that the scarring found during autopsy supported a traumatic brain injury was incorrect because this can only occur with "skull fractures, which Matthew did not have."

- Dr. Tlomak's testimony about the amount of force needed and her short-fall-distance opinions were wrong. He averred that a three-story fall would cause a "displaced skull fracture."
- A "sudden unexpected infant death in which the infant has been resuscitated and lives for several weeks or months may have identical autopsy findings to those of mechanical (impact) trauma."
- Dr. Tlomak's testimony about a prior resuscitated sudden infant death case showing different injuries than Matthew's was, in his view, irrelevant.

¶ 20 Cramer claims that Dr. Plunkett's testimony proves that the State's experts testified falsely at trial and violated his due-process rights. The trial court denied the motion without a hearing, ruling:

> *10 Dr. Plunkett's conclusions are no more than his opinions. The fact that his conclusions differ from those made by the State's witnesses does not establish that their testimony was false or misleading. Drs. Valvano and Tlomak were qualified to render the opinions they did, and Dr. Young offered his own opinion. There is no basis to conclude that Drs. Valvano or Tlomak made false or misleading statements because the defendant's postconviction expert takes issue with certain aspects of their testimony.

We now turn to Cramer's contentions on this appeal.

II.

A. *Alleged false testimony.*

¶ 21 As we have seen, Cramer claims Dr. Plunkett's opinions prove "the State presented demonstrably false and misleading testimony" and the trial court erred in summarily denying his motion. We, like the trial court, disagree.

¶ 22 The State may not use false testimony to get a conviction. *See Giglio v. United States,* 405 U.S. 150, 153–154, 92 S.Ct. 763, 31 L.Ed.2d 104 (1972), because this would, obviously, violate a defendant's right to due process, *Napue v. Illinois,* 360 U.S. 264, 269, 79 S.Ct. 1173, 3 L.Ed.2d 1217 (1959). To prove a due process violation, a defendant must show: "(1) that there was false testimony; (2) that the [State] knew or should have known it was false; and (3) that there is a likelihood that the false testimony affected the judgment of the jury." *United States v. Freeman,* 650 F.3d 673, 678 (7th Cir.2011). We review *de novo* a trial court's conclusion whether a defendant was denied due process because of the State's presentation of allegedly false evidence. *See State v. Burns,* 2011 WI 22, ¶ 23, 332 Wis.2d 730, 747, 798 N.W.2d 166, 174.

¶ 23 When a "defendant fails to allege sufficient facts in his motion to raise a question of fact, or presents only conclusionary allegations, or if the record conclusively demonstrates that the defendant is not entitled to relief, the trial court may in the exercise of its legal discretion deny the motion without a hearing." *Nelson v. State,* 54 Wis.2d 489, 497–498, 195 N.W.2d 629, 633 (1972).

¶ 24 Cramer has not shown that the State used false testimony. Both Dr. Valvano and Dr. Tlomak were qualified expert witnesses, and we do not understand Cramer to contend in his postconviction motion or on this appeal that they were not. *See* WIS. STAT. RULEE 907.02. Both physicians personally treated Matthew and saw evidence of his injuries first-hand. Both based their opinions on their training, experience, and knowledge. The fact that Dr. Plunkett (or Dr. Young for that matter) disagreed with their opinions does not make their testimony false. Indeed, Cramer's argument that Drs. Valvano and Tlomak testified *falsely* is tenuous at best. For example, Cramer argues that Dr. Valvano's testimony that: "There really is no controversy outside the courtroom. The American Academy of Pediatrics, pediatricians, neurosurgeons, it's well accepted that violently shaking a baby causes injury to that baby" is false given the medical literature on which he relies. The medical-literature controversy however, is not that "violently shaking a baby causes injury to that baby" but rather whether shaking alone, without some type of impact,

can cause the type of brain injury commonly associated with shaken baby syndrome in the past. *See State v. Edmunds,* 2008 WI App 33, ¶ 15, 308 Wis.2d 374, 385, 746 N.W.2d 590, 596 ("[A] significant and legitimate debate in the medical community has developed in the past ten years over whether infants can be fatally injured through shaking alone[.]"). As we have seen, Dr. Valvano testified that Matthew died from abusive head trauma, not shaken baby syndrome.

*11 ¶ 25 Cramer's other "false testimony claims" centered on: (1) short falls, and (2) that impacts with soft cushions do not leave external evidence of injury rest on red herrings. There is no history in the Record that Matthew fell—short or long. Moreover, Cramer's theory that short falls can cause injury, and that Matthew did not have any broken bones or other external injuries was also fully presented to the jury through Dr. Young's testimony and Cramer's cross-examination of Drs. Valvano and Tlomak. The jury believed the State's experts despite Cramer's expert's contrary opinions.

B. ***Alleged need for a Machner hearing.***

¶ 26 Cramer claims the trial court should have held a ***Machner*** hearing to determine whether his lawyer gave him constitutionally ineffective representation because his trial lawyer did not: (1) raise the lucid-interval in sudden-infant-death-syndrome situations; and (2) more effectively challenge the State's expert witnesses.

¶ 27 In order to show constitutionally ineffective representation, Cramer must show: (1) deficient representation; and (2) resulting prejudice. *See Strickland v. Washington,* 466 U.S. 668, 687, 104 S.Ct. 2052, 80 L.Ed.2d 674 (1984). To prove deficient representation, he must point to specific acts or omissions by his lawyer that are "outside the wide range of professionally competent assistance," *see id.,* 466 U.S. at 690, and to prove resulting prejudice, he must show that his lawyer's errors were so serious that he was deprived of a fair trial and reliable outcome, *see id.,* 466 U.S. at 687. We do not need to address both *Strickland* aspects if a defendant does not make a sufficient showing on either one. *See id.,* 466 U.S. at 697.

¶ 28 A circuit court must hold an evidentiary hearing on an ineffective-assistance-of-counsel claim only if the defendant " 'alleges sufficient material facts that, if true, would entitle the defendant to relief.' " *State v. Love,* 2005 WI 116, ¶ 26, 284 Wis.2d 111, 123, 700 N.W.2d 62, 68 (quoted source omitted). If the postconviction motion does not assert sufficient facts, or presents only conclusory allegations, or if the Record conclusively demonstrates that the defendant is not entitled to relief, the circuit court may deny the claim without a hearing. *Ibid.* We review *de novo* whether a defendant is entitled to an evidentiary hearing. *State v. Bentley,* 201 Wis.2d 303, 310, 548 N.W.2d 50, 53 (1996).

1. ***Lucid Interval.***

¶ 29 Cramer contends that his lawyer should have argued that there was a possibility that Matthew had a lucid interval because that, he claims, would have convinced the jury that he was not responsible for Matthew's death. There is, however, no evidence to support the lucid interval argument. Both Cramer and his wife denied that Matthew had had any prior accidental falls or trauma. Both Cramer and his wife said that Matthew had been healthy from birth and was acting normally the morning of the 911 call. Moreover, the defense's strategy was that the cause of Matthew's death was natural—a resuscitated sudden infant death. In other words, the trial lawyer's theory was that no one caused Matthew's death. If Cramer's lawyer would have added "lucid interval" to that defense, he would have had to argue that Matthew had an earlier accident or trauma. There is no evidence in the Record that Matthew had an earlier accident or trauma, and Cramer's appellate materials do not suggest otherwise. On our *de novo* review, the trial court did not err when it denied Cramer's request for a *Machner* hearing.

2. ***Challenging the State's experts.***

*12 ¶ 30 Cramer also argues his lawyer gave him constitutionally ineffective representation because he did not challenge the State's experts more vigorously on cross-examination. The Record forcefully belies this claim, however. As we have seen, Cramer's lawyer aggressively cross-examined the State's expert witnesses. Further, Cramer's lawyer presented the testimony of a strong defense expert witness, Dr. Young, who not only opined that Matthew's death had a "natural cause," but who also sharply criticized and contradicted the testimony of the State's experts. The fact that the jury did not credit Dr. Young's opinions,

does not make Cramer's lawyer constitutionally ineffective. Again, on our *de novo* review, the trial court appropriately denied Cramer's motion for a *Machner* hearing.

C. *Interest of Justice.*

¶ 31 Cramer asks us to reverse "in the interest of justice" because the jury did not hear Dr. Plunkett's testimony on shaken-baby-impact syndrome and lucid intervals. WISCONSIN STAT. § 752.35 controls discretionary reversals. That section provides:

> In an appeal to the court of appeals, if it appears from the record that the real controversy has not been fully tried, or that it is probable that justice has for any reason miscarried, the court may reverse the judgment or order appealed from, regardless of whether the proper motion or objection appears in the record and may direct the entry of the proper judgment or remit the case to the trial court for entry of the proper judgment or for a new trial, and direct the making of such amendments in the pleadings and the adoption of such procedure in that court, not inconsistent with statutes or rules, as are necessary to accomplish the ends of justice.

¶ 32 The "real controversy" here was what and who caused Matthew's death. The State's experts testified that Matthew died from abusive head trauma and Cramer's expert testified that Matthew died from sudden-infant-death syndrome, and attributed all of Matthew's internal injuries to Cramer's alleged efforts to resuscitate the child. Finding another expert after the trial who would have disagreed with the State's experts does not mean the real controversy was not tried. The jury heard the State's experts and Cramer's expert. It believed the State's. This is not one of those relatively rare situations where we should grant a new trial in the interest of justice. *See Vollmer v. Luety,* 156 Wis.2d 1, 11, 456 N.W.2d 797, 802 (1990). Indeed, Cramer's interest-of-justice contention is but the wine of his other arguments, which we have already rejected, repackaged in a new container. *See Mentek v. State,* 71 Wis.2d 799, 809, 238 N.W.2d 752, 758 (1976) ("We have found each of these arguments to be without substance. Adding them together adds nothing. Zero plus zero equals zero.").

***13** Judgment and order affirmed.

Parallel Citations

840 N.W.2d 138 (Table), 2013 WL 5612551 (Wis.App.), 2013 WI App 138

Footnotes

1 The Honorable Kevin E. Martens presided over Cramer's trial. The Honorable Jeffrey A. Wagner denied Cramer's motion for postconviction relief. The jury also found Cramer guilty of bail-jumping for violating a no-contact order by talking to his wife. He does not appeal that part of the judgment.

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The shaken baby syndrome

A clinical, pathological, and biomechanical study

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✓ Because a history of shaking is often lacking in the so-called "shaken baby syndrome," diagnosis is usually based on a constellation of clinical and radiographic findings. Forty-eight cases of infants and young children with this diagnosis seen between 1978 and 1985 at the Children's Hospital of Philadelphia were reviewed. All patients had a presenting history thought to be suspicious for child abuse, and either retinal hemorrhages with subdural or subarachnoid hemorrhages or a computerized tomography scan showing subdural or subarachnoid hemorrhages with interhemispheric blood. The physical examination and presence of associated trauma were analyzed; autopsy findings for the 13 fatalities were reviewed. All fatal cases had signs of blunt impact to the head, although in more than half of them these findings were noted only at autopsy. All deaths were associated with uncontrollably increased intracranial pressure.

Models of 1-month-old infants with various neck and skull parameters were instrumented with accelerometers and shaken and impacted against padded or unpadded surfaces. Angular accelerations for shakes were smaller than those for impacts by a factor of 50. All shakes fell below injury thresholds established for subhuman primates scaled for the same brain mass, while impacts spanned concussion, subdural hematoma, and diffuse axonal injury ranges. It was concluded that severe head injuries commonly diagnosed as shaking injuries require impact to occur and that shaking alone in an otherwise normal baby is unlikely to cause the shaken baby syndrome.

KEY WORDS • shaken baby syndrome • head injury • child abuse

THE term "whiplash shaken baby syndrome" was coined by Caffey[3] to describe a clinicopathological entity occurring in infants characterized by retinal hemorrhages, subdural and/or subarachnoid hemorrhages, and minimal or absent signs of external trauma. Because a nursemaid admitted that she had held several such children by the arms or trunk and shaken them, the mechanism of injury was presumed to be a whiplash-type motion of the head, resulting in tearing of the bridging veins. Such an injury was believed to be frequently associated with fatalities in infantile child abuse and has been postulated as a cause of developmental delay in survivors.[4,15]

While the term "shaken baby syndrome" has become well entrenched in the literature of child abuse, it is characteristic of the syndrome that a history of shaking in such cases is usually lacking.[12] Shaking is often assumed, therefore, on the basis of a constellation of clinical findings and on the computerized tomography (CT) picture of subarachnoid and subdural hematomas, particularly in the posterior interhemispheric fissure.[17] Because of the ambiguous circumstances of such injuries, medicolegal questions are particularly troublesome, and the neurosurgeon is often consulted to give an opinion as to whether the findings are consistent with child abuse or accidental injury.

This paper reviews all cases of the shaken baby syndrome seen at the Children's Hospital of Philadelphia (CHOP) between January, 1978, and March, 1985. To better study the mechanism of injury, autopsy results in all fatal cases were reviewed, and the biomechanics of this injury were studied in a series of infant models. Based on these observations, we believe that shaking alone does not produce the shaken baby syndrome.

Clinical Studies

Clinical Material and Methods

All reports submitted to the Suspected Child Abuse and Neglect team were reviewed. Since house officers

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TABLE 1
Initial clinical criteria for diagnosis of shaken baby syndrome

Diagnosis*	Cases		No. of Deaths
	No.	Percent	
retinal hemorrhage + SAH or SDH	29	60	5
retinal hemorrhage + SAH & SDH	10	21	5
bilateral chronic SDH	3	6	0
SAH &/or SDH & interhemispheric blood on CT	6	13	3
total	48	100	13

* SAH = subarachnoid hemorrhage; SDH = subdural hemorrhage; CT = computerized tomography.

TABLE 2
Best history in 48 cases of shaken baby syndrome

Etiology	Cases	
	No.	Percent
shaking only	1	2
fall or accidental blunt trauma	15	31
strike or fall plus shaking	10	21
strike only	3	6
trauma or shaking denied, caretakers in attendance	8	17
history unknown, caretakers not in attendance	10	21
cardiopulmonary resuscitation	1	2

TABLE 3
Trauma associated with shaken baby syndrome in 48 cases

Associated Trauma	Cases	
	No.	Percent
no evidence of blunt impact to head	18	37.5
no extracranial trauma	12	25.0
additional extracranial trauma	6	12.5
acute	3	6.25
old trauma only	3	6.25
evidence of blunt impact to head	30	62.5
skull fractures	12	25.0
cranial soft-tissue contusions	18	37.5
additional extracranial trauma	18	37.5
acute	15	31.25
old trauma only	3	6.25

and emergency room personnel are well trained in recognizing the clinical manifestations associated with this syndrome, it is considered that essentially all cases seen at CHOP are reported to this group.

Suspicion of shaking was based on history, clinical findings, and CT data. All subjects met the following criteria: presence of retinal hemorrhages with subdural and/or subarachnoid hemorrhages, bilateral chronic subdural hematomas, or a CT scan showing subdural or subarachnoid hemorrhages with interhemispheric blood. In addition, all patients were judged to have histories suggestive of child abuse or neglect; well-documented, witnessed accidental trauma was excluded. Histories were obtained from several interviews with caretakers by physicians, social workers, and in some cases law enforcement agents. Caretakers were routinely asked specifically about shaking.

Associated trauma data were obtained from physical examination, skull radiographs, CT scans, and skeletal surveys. All fatal cases were examined by the Philadelphia Medical Examiner, and pathology data were obtained from that office.

Results

Fifty-seven patients with suspected shake injury were identified. Of these, detailed clinical information was available in 48 cases. These patients ranged in age from 1 month to 2 years (mean 7.85 months). Thirty-one patients were male (65%). There were 13 fatalities (27%). Initial clinical criteria for diagnosis of the shaken baby syndrome are listed in Table 1. Thirty-nine patients (81%) had retinal hemorrhages plus subarachnoid and/or subdural hemorrhages. The remainder had bilateral chronic subdural hematomas (6%) or the above-mentioned CT findings without retinal hemorrhages (13%).

The most common presenting complaints were lethargy, breathing difficulty, irritability, poor feeding, and seizures. Best history is listed in Table 2; the most common histories were accidental blunt trauma (usually a fall) in 15 (31%) and blunt trauma plus shaking in 10 (21%); trauma and shaking were denied in eight (17%). In three cases (6%) the child was struck by the caretaker. In eight additional cases the history was unknown, usually because the child was left alone or with a babysitter. There were two cases (4%) with no history to explain the present findings, but both children were known to have been abused previously or subsequently. One case was associated with cardiopulmonary resuscitation (2%). In only one case was a history of shaking alone obtained; this child was reportedly shaken when she appeared to have difficulty in breathing associated with a respiratory infection.

Associated trauma observed clinically, radiographically, or at autopsy is listed in Table 3. The presence of scalp contusion, subgaleal or subperiosteal hemorrhage, and/or skull fracture was considered evidence of blunt impact to the head. Twelve cases (25%) had intracranial findings associated with the shaken baby syndrome alone, with no findings of associated blunt trauma to the head and no extracranial trauma. Six additional cases (13%) had the syndrome without signs of blunt head trauma but did have associated extracranial trauma. Thirty cases (63%) had findings of blunt impact to the head in addition to the intracranial findings of the shaken baby syndrome. Of these, 12 (25%) had skull fractures and 18 (38%) had significant cranial soft-tissue contusions. Most of the fractures were in the occipital or parieto-occipital region.

Clinical history, physical findings, hospital course, intracranial pressure (ICP, when measured), and pathological findings of the 13 fatalities are listed in Tables

TABLE 4

*Clinical and pathological findings in 13 fatal cases of shaken baby syndrome**

Factor	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8	Case 9	Case 10	Case 11	Case 12	Case 13
age (mos)	24	7	3	22	11	9	8	5	10	13	24	4	19
sex	F	M	M	M	F	F	F	M	F	M	M	M	F
history													
fall or hit	+		+	+	+			+	+	+	+	+	+
shaking			+								+		+
trauma denied													
unknown		+				+	+						
initial examination													
unresponsive	+	+	+	+	+	+	+	+	+	+	+	+	+
retinal hemorrhages	+	+		+			+	+	+	+	+	+	
cranial impact	+			+						+	+	+	+
extracranial trauma	+			+	+						+	+	
intracranial pressure	↑↑	NM	↑↑	↑↑	↑↑	NM	↑↑	NM	↑↑	NM	↑↑	↑↑	↑↑
survival time (days)	2	2	7	2	3	2	2	1	1	1	4	1	1
pathology													
cranial contusions	+	+	+	+	+	+	+	+	+	+	+	+	+
skull fracture(s)	++			++	++							+	+
subdural hematoma	+	+	+	+	+	+	+	+	+	+	+	+	+
subarachnoid hemorrhage	+	+	+	+	+	+	+	+	+	+	+	+	+
hemispheric contusions	+	+	+			+		+					+
white matter tears	+					+			+				
diffuse brain swelling	+	+	+	+	+	+	+	+	+	+	+	+	+

* ↑↑ = increased; NM = not measured; + = factor present; ++ = severe.

4 and 5. Mean age in this group was 12.23 months; 54% were male. All of these children arrived at the hospital in an essentially unresponsive state, and all died from the effects of uncontrollably increased ICP associated with massive brain swelling. In only one case was a subdural hematoma thought to be of significant size to warrant surgical intervention, and drainage was ineffective in controlling elevated ICP.

Pathological examination showed that all of the children who died had evidence of blunt head trauma. Eight had soft-tissue contusions and five had contusions and skull fractures. In seven cases, however, impact findings were noted only at autopsy, and had not been apparent prior to death. All fatal cases had subdural and subarachnoid bleeding. Focal cerebral contusions and lacerations occurred in six. Microscopic examination was performed in three cases and showed corpus callosum hemorrhages, cortical laminar necrosis, or white matter hemorrhages. All children had diffuse and usually massive brain swelling.

Biomechanical Studies

Whole Infant Models

To test the hypothesis that infants are particularly susceptible to injury from shaking because of a relatively large head and weak neck, we constructed models of 1-month-old infants that were implanted with an accelerometer to measure the results of shaking or impact manipulations. Since the mechanical properties of the infant neck have not been studied, three models were built with different neck structures in order to include the range of limiting conditions that might exist in the live infant. Both a fixed center of rotation with zero resistance (hinge model) and moving centers of rotation with low and moderate resistance (rubber neck models) were tested.

Experimental Methods

The heads and bodies of the models were adapted from Just Born dolls. Head circumference was 36 cm, coronal width was 10 cm, anteroposterior diameter was 10.75 cm, and height from vertex to base (calculated from a line drawn from chin to caudal occiput) was 9.0 cm; values were comparable to human infants. Brain weight for an infant of this size was assumed to be 500 gm.[1] The ideal weight of the head was estimated by balance-weight measurements of several infants with an average age of 1 month, and was 770 to 870 gm. The heads of the models were tightly filled with cotton, with water added until the desired weight range was reached. The water was absorbed by the cotton and distributed so that no sloshing of the contents occurred. The heads were reweighed after neck insertion and sealing and at the end of all experiments.

Neck length from the skull base to the T-1 vertebra

TABLE 5

Summary of findings in 13 fatal cases of shaken baby syndrome

Factor	Finding
age (mos)	
mean	12.23
range	3–24
sex M/F	7/6
history	
fall or hit (three with shaking)	10
unknown	3
initial examination	
unresponsive	13
retinal hemorrhages	9
cranial impact	6
extracranial trauma	5
intracranial pressure	
measured, unable to control	9
not measured	4
survival time (days)	
range	1–7
mean	2.2
pathology	
cranial contusions	13
skull fractures(s)	5
subdural hematomas (one requiring surgery)	13
subarachnoid hemorrhage	13
unilateral	3
diffuse	3
multifocal	7
hemispheric contusions	6
diffuse, multiple	3
focal, coup-contrecoup	3
white matter tears	4
gross	2
microscopic	2
diffuse brain swelling (11 with herniation evident)	13

TABLE 6

Mean acceleration and time course of shakes and impacts in all models

Manipulation	No.	Peak Tangential Acceleration (G)	Time (msec)	Angular Velocity (radians/sec)	Angular Acceleration (radians/sec^2)
shakes	69	9.29	106.6	60.68	1138.54
impacts	60	428.18	20.9	548.63	52,475.70

TABLE 7

Effects of neck condition and "skull" on mean peak tangential acceleration and time course of shakes and impacts

Variant	Shakes		Impacts	
	Acceleration (G)	Time (msec)	Acceleration (G)	Time (msec)
hinge neck	13.85	92.7	423.42	18.6
flexible rubber neck	5.70	93.3	427.78	21.4
stiff rubber neck	7.02	130.5	433.33	22.8
skull	9.86	107.4	436.12	20.2
no skull	8.89	103.5	427.04	21.6

TABLE 8

Effect of impact surface on mean peak tangential acceleration and time course

Surface of Impact	Acceleration (G)	Time (msec)
padded surface	380.60	24.22
metal bar	489.51	17.13

was measured from lateral neck films of several normal infants with an average age of 1 month and ranged from 3.5 to 4.5 cm; all models were therefore given neck lengths of 4.0 cm. Necks were embedded in Castolite resin* superiorly, which was also used to seal the head. The interior part of the neck was secured in dental stone.† The stuffed body was then replaced around the dental stone "thorax," with lead weights added as necessary to the thorax to reach a total body weight of 3 to 4 kg. Arms and legs were not weighted, so the slightly low total weight for age reflects an attempt to approximate trunk:head weight ratios.

Model 1 had a hinge neck made from a 360° steel hinge, 3.6 cm in width, placed in the horizontal plane to allow complete anteroposterior angulation of the head. The center of rotation was 3.3 cm below the estimated level of the skull base (approximating at the C-6 vertebral level). Model 2 had a 1.9-cm diameter hollow rubber neck with a 0.8-cm lumen. This neck did not support the weight of the head in the upright position but did not kink when the head was allowed to fall unsupported. Model 3 had a 2.9-cm rubber neck with a 1.2-cm lumen. This neck was able to support the head in the vertical position but allowed full passive movement of the head. In all models, head motion was limited in the anteroposterior direction by the occiput striking the upper back and the chin striking the chest.

To test for the effect of the deformability of the model heads on impact, all models were tested with and without an external "pseudoskull" made from thermoplastic.‡ This "skull" was 1/8 in. thick and was molded to the occipital, parietal, temporal, and posterior frontal areas, with the facial area uncovered. The "skulls" weighed 170 to 200 gm.

Data were recorded from a piezoelectric accelerometer§ embedded in a small piece of thermoplastic and attached to the vertex in a coronal plane through the

* Resin manufactured by Buehler Ltd., Evanston, Illinois.

† Dental stone, Glastone Type IV, manufactured by Ransom and Randolph Co., Toledo, Ohio

‡ Polyform thermoplastic manufactured by Rolyan Medical Products, Menomonee Falls, Wisconsin.

§ Accelerometer manufactured by Endevco Corp., San Juan Capistrano, California.

15

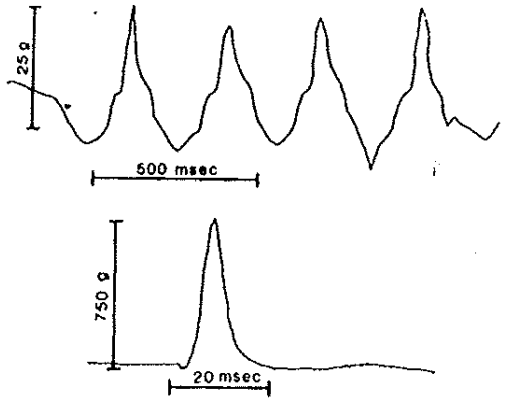

FIG. 1. Representative tangential acceleration traces for infant models undergoing shake *(upper)* and impact *(lower)* manipulations. While manipulations of the infant models were performed as described, with a series of shakes followed by an impact, the magnitude of the impact accelerations was so much greater than that associated with the shakes that different scales are used to display the respective acceleration traces.

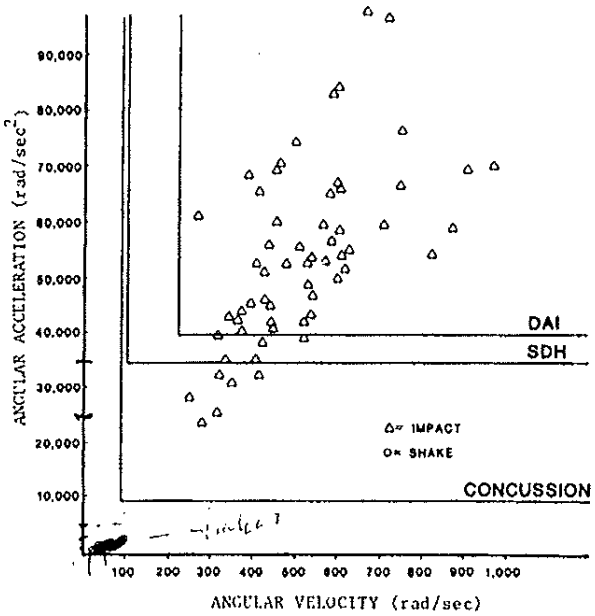

FIG. 2. Angular acceleration versus angular velocity for shakes and impacts, with injury thresholds from primate experiments scaled to 500-gm brain weight. DAI = diffuse axonal injury; SDH = subdural hematoma.

center of the neck. Each model was subjected to repetitive violent shaking, allowing the head to travel its full excursion several times, by adult male and female experimenters. The models were held by the thorax facing the experimenter and were shaken in the anteroposterior plane, since this is the motion most commonly described in the shaken baby syndrome. At the end of each series of shakes the occiput was impacted against either a metal bar or a padded surface. Each model was tested at least 20 times. Acceleration traces were amplified and recorded.‖

Angular accelerations were calculated from the measured peak tangential accelerations by using C-6 as the center of rotation in all cases. Angular velocity was calculated as the time integral of the acceleration curve. Translational forces were assumed to be minimal.

Results

The data were collected from 69 shaking episodes ("shakes") and 60 "impacts." Typical tangential acceleration traces for shake and impact manipulations are shown in Fig. 1. The criterion for significant difference was $p < 0.01$ in all cases.

Shakes Versus Impacts. Angular acceleration and angular velocity for each shake and impact are shown in Fig. 2. Mean peak tangential acceleration for 69 shaking episodes was 9.29 G; mean peak tangential acceleration for 60 impacts was 428.18 G (Table 6). The accelerations due to impact are significantly greater than those obtained by shaking ($p < 0.0001$); on the average, impact accelerations exceed shake accelerations by a factor of nearly 50 times. Mean time interval for shakes was 106.6 msec and for impacts was 20.9 msec. This difference is significant at the $p = 0.001$ level.

Effects of Neck Condition. Mean tangential accelerations and time courses for shakes and impacts for each neck condition are presented in Table 7. There is no significant difference between the hinge neck, the flexible rubber neck, and the stiff rubber neck in the mean acceleration resulting from impacts (423.4, 427.8, and 433.3 G, respectively) or in the mean time course (18.6, 21.4, and 22.8 msec, respectively). With shakes, the more flexible hinge neck is associated with higher accelerations (mean 13.85 G) than the two rubber neck models (mean 5.7 and 7.0 G) ($p < 0.001$). There is an inverse relationship between neck stiffness and time duration of a shake: the stiff rubber neck was associated with a longer time course than the more flexible rubber neck (130.5 msec and 93.3 msec, respectively) ($p < 0.001$).

Effects of "Skull." The presence of a hard thermoplastic "skull" did not change the magnitude or time course of accelerations associated with shaking of the models. The acceleration magnitude and time course were also unchanged when the models were impacted. These data are shown in Table 7.

Effects of Impact Surface. Impact against a padded surface was associated with significantly smaller acceleration (mean 380.6 G) and longer time course (mean 24.22 msec) than that against a metal bar (mean 489.5 G and 17.13 msec) ($p < 0.001$). Data are shown in Table 8.

‖ Shock amplifier, Model 2740 A, and pulse memory unit, Model 2743, manufactured by Endevco Corp., San Juan Capistrano, California.

Discussion

Clinical head injury can be classified into two major categories according to the distribution of pathological damage, whether focal or diffuse.[10] Such a distinction is important for treatment and prognosis, as well as for establishing the biomechanical conditions necessary to produce a given injury type. It has been established both experimentally and clinically that most focal injuries are associated with impact loading, resulting in contact phenomena, while diffuse injuries are associated with impulsive loading conditions resulting from acceleration-deceleration phenomena.[6] Damage to the brain occurs as a result of these biomechanical forces and from the secondary effects of ischemia due to altered autoregulation or brain swelling.

The shaken baby syndrome has been postulated to result from the effects of nonimpact acceleration-deceleration forces. It has been suggested that the back and forth movement of the head alone is sufficient to cause tearing of bridging veins, resultant subdural hematomas, and death.[8,13] The relatively large size of an infant's head, weakness of the neck musculature, softness of the skull, relatively large subarachnoid space, and high water content of the brain have been postulated to contribute to the susceptibility of shaking injuries in infants.[4,14]

While shaking alone has been considered sufficient to cause a fatal injury, the usual lack of history of the true mechanism of injury in these cases has hampered accurate clinicopathological correlations. It is of interest, however, that in a recent series of fatal cases of infantile head injuries from suspected child abuse,[5] white matter tears were found similar to those described by Lindenberg and Freytag[11] in blunt trauma in infancy. In addition, lesions in the distribution typical of diffuse axonal injury, like those found in adult head injury and in subhuman primates subjected to high acceleration-deceleration injury,[7] were described in some cases. In fact, at least one of Caffey's original cases[3] included "lacerations of the cerebral parenchyma." Shaking alone was the presumed mechanism of these injuries.

As experience has accumulated in experimental angular acceleration injury it has become clear that, besides the magnitude of the acceleration, another important biomechanical factor influencing injury type is the time interval over which the acceleration occurs. Thus, large angular accelerations occurring over shorter time periods tend to result in subdural hematoma, while longer intervals are associated with diffuse axonal injury.[6] A tolerance scale relating these two factors to resultant injury has been developed for the subhuman primate by Thibault and Gennarelli.[16] Values above certain critical limits result in a particular type of injury such as concussion, subdural hematoma, or diffuse axonal injury. When such a curve is scaled for the brain mass of an infant the size of our models, it can be seen that the angular acceleration and velocity associated with shaking occurs well below the injury range, while the values for impacts span concussion, subdural, and diffuse axonal injury ranges (Fig. 2). This was true for all neck conditions with and without skulls. A padded surface decreases the magnitude of acceleration and lengthens the time course to some extent, but these impacts also fall in the injury range.

These results are consistent with the observation that the fatal cases of the shaken baby syndrome in this series were all associated with evidence of blunt impact to the head. This preponderance of blunt trauma has also been found in at least one other series of nonaccidental head trauma in childhood in which the mechanism of injury was investigated.[9] It is of interest that in more than half of our fatal cases, no evidence of external trauma was noted on the initial physical examination, which helped to contribute to the diagnosis of "shaken baby syndrome." Skull fractures and scalp contusions were found at autopsy, however, most often in the occipital or parieto-occipital region. In addition, several babies had parenchymal lesions in a distribution consistent with diffuse axonal injury.[11]

While some reports on the shaken baby syndrome mention brain swelling, in most reports the subdural collections themselves have been postulated as the cause of death. In this series, all fatalities were consequent to uncontrollable brain swelling, and it is clear that drainage of the small collections present would have been useless in controlling the ICP. The problem of acute brain swelling is particularly common in the pediatric population, and its cause is poorly understood.[2] Whether high accelerations in the anteroposterior direction have some particular association to this complication remains to be investigated.

It is our conclusion that the shaken baby syndrome, at least in its most severe acute form, is not usually caused by shaking alone. Although shaking may, in fact, be a part of the process, it is more likely that such infants suffer blunt impact. The most common scenario may be a child who is shaken, then thrown into or against a crib or other surface, striking the back of the head and thus undergoing a large, brief deceleration. This child then has both types of injury — impact with its resulting focal damage, and severe acceleration-deceleration effects associated with impact causing shearing forces on the vessels and parenchyma. Unless a child has predisposing factors such as subdural hygromas, brain atrophy, or collagen-vascular disease, fatal cases of the shaken baby syndrome are not likely to occur from the shaking that occurs during play, feeding, or in a swing, or even from the more vigorous shaking given by a caretaker as a means of discipline.

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Article

A DAUBERT ANALYSIS OF ABUSIVE HEAD TRAUMA/SHAKEN BABY SYNDROME

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Abusive Head Trauma (AHT) has been known over the years by multiple terms-- "Whiplash Shaken Baby Syndrome," [1] "Whiplash Shaken Infant Syndrome," "Shaken Impact Syndrome," "Inflicted Childhood Neurotrauma," "Non-Accidental Trauma," and others. To the lay public, it is most commonly referred to, or recognized as "Shaken Baby Syndrome" (SBS). Irrespective of the vernacular, [2] AHT has long been recognized as a clinically valid medical diagnosis. [3] However, recent legal literature, [4] public media, [5] ***506** and court decisions have called into question the foundation, and consequent validity, of AHT/SBS as a valid medical diagnosis. [6]

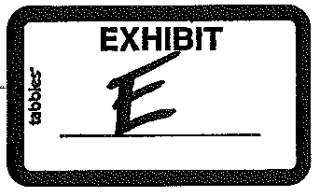
Because of the diagnosis' direct translation and impact in the legal arena, some have gone so far as to champion the cause of its invalidation under philosophical banners of "protection of the innocent" and "justice." [7] Broad assertions and generalizations have been proffered, such as: "the scientific underpinnings of SBS have crumbled over the past decade;" [8] or the medical research underlying SBS is a "flawed science" [9] predicated upon "circular reasoning," "data gaps," and "inconsistency of case definition." [10] Additionally, it has been asserted that "as technology and scientific methodology advanced, researchers questioning the basis for SBS reached a ***507** critical mass." [11]

Despite the assertions, what has not been published thus far is a detailed, critical analysis of the medical literature surrounding AHT, and not only whether that literature meets the Trilogy (Daubert, Joiner, and Kumho) criteria for admissibility of scientific evidence/testimony, but whether that literature is "flawed" and consequently not predicated upon sound scientific and medical principles. [12] Part I of this paper shall examine the Trilogy (Daubert, Joiner, and Kumho) criteria for admissibility of expert testimony/evidence, and the medical and legal quests for sound scientific evidence. Part II of this paper shall explore the issues surrounding the medical diagnosis of AHT. Specifically, we shall review basic statistical principles utilized in critical evaluation of medical/scientific literature and then critically analyze the medical literature involving some of the more common injuries [13] associated with AHT. Finally, Part III of this paper shall assess not only whether the medical literature suffices under Daubert, Joiner, and Kumho scrutiny, but shall briefly examine the contemporary legal ***508** issues surrounding admissibility of AHT testimony and proffer some solutions for those issues.

I. The Trilogy: Daubert, Joiner, and Kumho

A. Daubert v. Merrell Dow Pharmaceuticals, Inc.

For many years in the twentieth century, expert testimony on novel scientific evidence was admissible only if the opinion offered was based on a "well-recognized scientific principle or discovery . . . [that was] sufficiently established to have gained general acceptance in the particular field in which it belongs." [14] That standard, enunciated in Frye v. United States, was also known



as the "general acceptance" test. [15] In 1993, with the Supreme Court's ruling in Daubert v. Merrell Dow Pharmaceuticals, Inc., that standard changed. [16]

In Daubert, the Supreme Court evaluated the propriety of a lower court's ruling excluding certain expert testimony in a tort liability case. [17] In the case, Petitioners Jason Daubert and Eric Schuller were minor children born with serious birth defects. [18] They and their parents had sued the respondent, Merrell Dow Pharmaceuticals, alleging that the birth defects were caused by the mother's ingestion of Merrell Dow's drug, Bendectin (an antinausea medication). [19] The Petitioners sought to proffer expert testimony. [20] The district court, applying the "general acceptance" test of Frye, denied the admissibility of the petitioner's expert testimony, and granted summary judgment for the respondent. [21] To settle the divisions among the lower courts regarding the proper standard for the admission of expert testimony, the Supreme Court granted ***509** certiorari. [22]

The Court held unanimously that the Frye test had not survived. [23] With regards to the admissibility of expert testimony/ evidence, the Court held that Federal Rules of Evidence (FRE) 702 governs, not Frye. [24] The Daubert court held the text of FRE 702, its drafting history, and prior case law [25] mandated a "liberal" and "relaxed" approach to the admission of expert opinion testimony. [26] The inquiry into admission of expert testimony/evidence was within the province of the trial judge. While the trial judge's inquiry was to be a "flexible one," [27] the Daubert court required trial judges to ensure "that any and all scientific testimony or evidence admitted is not only relevant, but reliable." [28]

With regards to reliability, the Daubert Court stated that "[t]he subject of an expert's testimony must be 'scientific . . . knowledge.'" [29] The Court noted there were definitional differences between science and law on "reliability." [30] But the Court went on to state that "evidentiary reliability will be based upon scientific validity." [31] The Court enunciated four factors a trial judge could consider in the preliminary assessment of whether proposed testimony was scientifically valid:
1) whether a theory or technique could be (and had been) tested--also known as "falsifiability" or "testability";

2) whether the theory or technique had been subject to peer review and publication;

3) whether there was a known or potential rate of error; and

***510** 4) whether there was general acceptance in the relevant scientific community. [32]

The Court remarked that these factors were not a "definitive checklist or test," but merely factors for consideration in a trial judge's overall assessment. [33] The Court concluded by stating, "[t]he inquiry envisioned by Rule 702 is, we emphasize, a flexible one . . . . The focus, of course, must be solely on principles and methodology, not on the conclusions that they generate." [34]

With regards to relevance, the Court explained that expert testimony cannot assist the trier of fact in resolving a factual dispute, as required by Rule 702, unless the expert's theory is "sufficiently tied to the facts of the case." [35] The Court remarked, "Rule 702's 'helpfulness' standard requires a valid scientific connection to the pertinent inquiry as a precondition to admissibility." [36]

B. General Electric Co. v. Joiner

In General Electric Co. v. Joiner, the Court, in expanding upon the Daubert standard, examined and decided two additional, significant issues regarding the admissibility of scientific expert testimony. [37] First, the Court determined the appropriate standard for appellate review of a trial court's determination of admissibility of scientific expert testimony. After establishing an

abuse of discretion standard for appellate review, [38] the Court went on to examine a more important issue of whether existing scientific evidence can be generalized to address specific causal relationships. [39]

In Joiner, the plaintiff asserted that exposure to polychlorinated biphenyls had promoted the development of his small-cell lung *511 cancer. [40] The plaintiff argued that collective consideration of epidemiologic studies (which, when considered individually and separately, were equivocal), demonstrated a causal relationship. [41] In rejecting this argument, the Court determined the lower court had not abused its discretion in excluding this scientific testimony because there was no logical nexus between the methodology employed by the expert and the expert's conclusion. [42] The Court stated:

> Trained experts commonly extrapolate from existing data. But nothing in either Daubert or the Federal Rules of Evidence requires a district court to admit opinion evidence that is connected to existing data only by the ipse dixit of the expert. A court may conclude that there is simply too great an analytical gap between the data and the opinion proffered. [43]

C. Kumho Tire Co. v. Carmichael

In Kumho Tire Co. v. Carmichael, the Court examined the issue of the extent of a trial court's "gate-keeping" obligation. [44] Did it extend only to expert testimony based upon "scientific" knowledge or did it also apply to expert testimony based on "technical" and/or "other specialized knowledge"? In unanimously holding that a trial court's "gate-keeping" obligation extended to ALL expert testimony, the Court remarked that Federal Rule of Evidence 702 "makes no relevant distinction between 'scientific' knowledge and 'technical' or 'other specialized' knowledge." [45] Assurance of reliability of expert testimony, whether "scientific" or based upon "technical or other specialized knowledge," was still required. [46]

*512 In grappling with this issue, the Court remarked that there will be witnesses "whose expertise is based purely on experience. . . ." [47] The Court anticipated there would be times when such proffered expert testimony would have to be excluded because the expert's field lacks reliability. [48] But other than citing astrology and necromancy as such excludable disciplines, the Court gave no specific guidance on how a trial court could come to such a conclusion. [49] Instead, the Court proffered general guidance--the "intellectual rigor" test. [50]

The Court noted that the four Daubert factors "may or may not be pertinent [: it will all depend] on the nature of the issue, the expert's particular expertise, and the subject of his testimony." [51] The Court concluded that a trial court must exercise its gate-keeping obligation so that the expert, whether relying on "professional studies or personal experience, . . . [will, when testifying, employ] the same level of intellectual rigor" that the expert would use outside the courtroom when working in the relevant discipline. [52] In the words of one legal scholar:

> The Court seems less absorbed in epistemological issues, in formulating general rules for assessing reliability, or in fleshing out *513 the implications of its having singled out testability as the preeminent factor of concern. It appears less interested in a taxonomy of expertise and more concerned about directing judges to concentrate on "the particular circumstances of the particular case at issue." This flexible, nondoctrinaire approach is faithful to the intention of the drafters of the Federal Rules of Evidence . . . . [53]

Essentially, for physicians, the Court's decision in Kumho "tethered" the admissibility standard of expert testimony to the standards of medical practice. [54]

D. The Quest for Sound "Scientific Evidence/Testimony"

"Science is simply common sense at its best; that is, rigidly accurate in observation and merciless to a fallacy in logic." [55]
Thomas Henry Huxley

1. The Legal Perspective

The objective of law is justice. [56] Yet, justice is not merely the search for dispassionate truth, but dispassionate truth that results in fair and equitable decisions. [57] As the age of science has flourished, science and medicine have increasingly permeated the law and played crucial roles in the courtroom. [58]

In criminal law, the emergence of DNA sampling has resulted in the exoneration of those who were unjustly convicted and has ***514** provided greater confidence in the reliability of future convictions. [59] In tort law, courts are constantly confronted with causation or risk of injury determinations, which rely heavily on scientific or medical information. [60] In patent law, cases are heavily immersed in, and decisions frequently hinge upon, technical or scientific information. [61] And, in recent years, the Supreme Court has examined scientific and medical issues ranging from the propriety of statistical sampling techniques in the undercounting of certain identifiable groups on the decennial census, [62] to the constitutionality of a state psychopath statute, [63] to the constitutional question of whether the right to liberty in the Due Process Clause of the Fourth Amendment affords citizens a "right to die." [64]

As our scientific world has grown increasingly complex, courts have become increasingly wary of exposing juries to such potentially confusing evidence. Additionally, courts have recognized the inherent weight and persuasiveness the designation of "scientific evidence" can have in the minds of triers of fact. Bolstering that concern, some research suggests that as evidence becomes more complex and difficult to comprehend, jurors shift their focus to "peripheral indicia of reliability such as the expert's qualifications or demeanor," and are more likely to defer to the expert's opinion rather than forming their own. [65] This deference to ***515** scientific evidence has been labeled by some courts as the "aura of infallibility." [66] Furthermore, a few recent case reports of wrongful convictions have exacerbated those concerns of juror over-reliance on "scientific evidence." [67]

Nevertheless, in hopes of diminishing the admission of unreliable testimony, courts and legal scholars, both domestic and international, have endeavored to define sound scientific evidence. The Daubert Court stated:
The adjective "scientific" implies a grounding in the methods and procedures of science. . . . "Science is not an encyclopedic body of knowledge about the universe. Instead, it represents a process for proposing and refining theoretical explanations about the world that are subject to further testing and refinement" . . . . Proposed testimony must be supported by appropriate validation--i.e., "good grounds," based on what is known. [68]

In the words of one learned commentator, evidence is scientifically valid if "it results from sound and cogent reasoning." [69] Other scholars, echoing the Court's decisions in Daubert and Kumho state, "[i]t is how conclusions are reached, not what the conclusions are, that makes them 'good science.'" [70] In the words of the Honorable Stephen Breyer, Associate Justice of the Supreme Court:
***516** The search is not a search for scientific precision. . . . A judge is not a scientist, and a courtroom is not a scientific laboratory. But consider the remark made by the physicist Wolfgang Pauli. After a colleague asked whether a certain scientific paper was wrong, Pauli replied, "That paper isn't even good enough to be wrong!" Our objective is to avoid legal decisions

that reflect that paper's so-called science. The law must seek decisions that fall within the boundaries of scientifically sound knowledge. [71]

In the United Kingdom, the Law Commission recently proposed reformation of English Law with regards to admissibility of expert scientific evidence. [72] After a comprehensive review of the topic, the Commission found the Daubert court's analysis and conclusions regarding the admissibility of expert scientific testimony and evidence to be cogent, sound, and, ultimately, convincing. [73] Noting that many judges in England and Wales were already making admissibility decisions based upon the Daubert standard, the Commission recommended formal adoption of Daubert's "gate-keeping" role for a trial judge and Daubert's validity-based (reliability and relevance) admissibility test for expert scientific evidence. [74]

Although many have judged the trilogy (Daubert, Joiner and Kumho) to be a laudable attempt to bridge the treacherous crosscurrents of science and law, numerous issues regarding the determination of "sound scientific testimony" have remained unanswered. For example, with regards to the "analytical gap" between research data and expert opinion addressed in Joiner, [75] what is a sufficient amount and quality of evidence an expert may rely upon in bridging that "gap" in forming his/her opinion? Are medical textbooks (which are essentially expert treatises) authoritative references upon which experts may rely in forming their opinions? With regards to the "intellectual rigor" test of Kumho, what will be the applicable standard of professional practice to apply when, as often occurs in medical practice, multiple disciplines ***517** are involved? Who determines the applicable standard of professional practice? Individual experts? National organizations? Additionally, some have echoed concerns about the onerous burden Daubert's gate-keeping requirements have placed on the single trial judge. [76] As the Honorable Judge Alex Kozinski of the Ninth Circuit Court of Appeals stated:

> Our responsibility, then, unless we badly misread the Supreme Court's opinion, is to resolve disputes among respected, well-credentialed scientists about matters squarely within their expertise, in areas where there is no scientific consensus as to what is and what is not "good science," and occasionally to reject such expert testimony because it was not "derived by the scientific method." Mindful of our position in the hierarchy of the federal judiciary, we take a deep breath and proceed with this heady task. [77]

Empirical evidence has substantiated Judge Kozinski's concerns. In a 2001 survey of 400 state court judges, 96% of the judges failed to demonstrate even a basic understanding of two of the four Daubert criteria. [78] When assessing the concept of "falsifiability," a principle specifically enunciated in Daubert, 96% of ***518** the judges lacked even a basic understanding of this core scientific concept. [79] When asked to comment on the value of Daubert to their decision-making process, only 55% of judges found Daubert to provide a "great deal" of value. [80] Consequently, the researchers concluded that "[t]he survey findings strongly suggest that judges have difficulty operationalizing the Daubert criteria and applying them . . . ." [81]

Expectedly, the courts have grappled with confusion and responded with variable and inconsistent decisions. Some courts have attempted to reduce determinations of sound scientific evidence to "simple all-or-nothing rules, such as . . . doubling . . . the background rate of disease as proof of causality." [82] Some have required peer-reviewed studies [83] or statistical data [84] prior to admitting expert testimony. Some have dismissed case reports as non-scientific, [85] whereas other courts have given them significant weight. [86] Finally, some courts have disallowed expert testimony when such reliance was based primarily upon "animal studies [, have] cautioned against extrapolation of dosage levels, and [have] objected to generalization across similar substances." [87]

Whereas courts once greeted scientific evidence and testimony with deferential respect and relative trust, recent empirical data demonstrates that the legal pendulum has swung the other way. An ***519** "analysis by the Rand Corporation of a sample of 399

published and unpublished federal district court decisions" demonstrated a more restrictive approach by federal courts to the admissibility of scientific testimony and a shift "toward excluding proffered scientific and technical evidence." [88] Additionally, a recent survey of federal judges and attorneys by the Federal Judicial Center "confirmed a shift toward more demanding standards for admissibility" of scientific testimony and evidence. [89] In the words of one learned commentator, "[t]he courts appear to be asserting standards that they attribute to the medical profession, but that are inconsistent and sometimes more demanding than actual medical practice." [90]

2) The Medical Perspective

If the objective of law is justice, then the objective of medicine is to care for the patient. To truly understand the medical perspective, one must understand and accept the canon that medicine is inherently, by its nature, an inexact science. [91] There are aspects of medicine (for example laboratory research), which are more scientific in nature. But the fields of medicine that deal with direct patient interaction, also known as clinical medicine, are not exclusively scientific. The human interaction inherently introduces variables (such as the nuances of effective communication and an individual's behavioral, social, economic, and cultural norms and biases) that are not readily reducible to empirical scientific data and most certainly affect the outcome. The medical provider's judicious interplay of the human variable with the scientific data of the ***520** human body is what has been termed by many as the art [92] of clinical medicine. [93]

It is important to understand that the designation of an "art" is not a relegation to imprecision or lack of reliability. On the contrary, clinical medical decision-making is grounded in the roots of the scientific method. As Dr. Mark McClellan, Co-Chair of Institute of Medicine's 2007 Annual Meeting, stated, "[physicians'] education includes the scientific basis of health and disease. They have been trained to use scientific literature to compare alternative approaches to diagnosis and treatment. They do their best to stay up-to-date through reading and conferences." [94] Additionally, physicians receive basic training on statistical analysis, often apply those principles to critically evaluate the medical literature, and sometimes pursue advanced degrees in statistical expertise (like biostatistics or epidemiology).

While the cognitive underpinnings of the diagnostic process are rational and scientifically sound, ultimately, "[a]ll diagnostic hypotheses represent probabilistic judgments . . . that have variable probabilities of being correct." [95] Furthermore, physicians are as susceptible as anyone to biases, preconceptions, or "intrusions of emotion," any or all of which can influence clinical judgment and actions. [96] Physicians can, and do, avoid, or at least minimize, errors in cognition by maintaining awareness of the pitfalls of heuristics, and how personal biases and emotional temperature can affect them. [97]

***521** Physicians have continually reflected upon the clinical decision-making process, repeatedly assessing its cogency and need for improvement. [98] As technologic advancements in medical informatics occurred in the 1970s and 1980s, large volumes of medical literature were synthesized into computer indices and became available for large-scale statistical analysis. [99] This bred a new type of medical evidence, the systematic review. [100] On the heels of these technologic innovations, and the consequent ability to conduct comprehensive reviews of large volumes of medical literature, the Evidence-Based Medicine (EBM) movement came afoot. [101]

EBM has been characterized by one of its pioneers, Dr. David Sackett, as the "conscientious, explicit, and judicious use of current best evidence in making decisions about individual care." [102] Dr. Harvey Fineberg, President of the Institute of Medicine, recently stated that, "[t]he central notion in EBM [is] the importance of integrating individual clinical expertise with the best available external evidence." [103] This will provide "a helpful framework for providers to navigating uncertainty inherent in patient care." [104] In fact, most healthcare providers strive to be "evidence-based" in their ***522** practice. [105]

Despite an increased focus on "evidence basis" in their practice, "studies repeatedly show marked variability in what healthcare providers actually do in a given [clinical] situation." [106] Many had hoped that EBM would be the panacea to the judicial pains over medical practice guidelines and interpretation of medical evidence. However, as lingering controversies between reputed medical bodies [107] demonstrate, it has not been that panacea. [108] Additionally, there are some areas of medicine, where the evidence is so sparse, that EBM simply cannot be instructive either for Medicine or Law. [109]

Ultimately, the physician must sagely balance his scientific knowledge, underscored by statistical data, his emotional temperature and potential biases, and the myriad complexities that make up the "human" variable. "Statistics cannot substitute for the human being before you; statistics embody averages, not individuals. Numbers can only complement a physician's personal experience . . . ." [110] That is the "Art" of Clinical Medicine. Explicit evidence is only a portion of what physicians do.

II. Abusive Head Trauma as a Medical Diagnosis

"Those who cannot remember the past are condemned to repeat it." [111]
George Santayana

*523 A. History

Much of what we currently know about AHT is the result of decades of meticulous, tireless work by physicians from various disciplines from all over the world. [112] Many of these historical clinicians did not have the benefit of advanced laboratory or radiographic techniques such as coagulation (clotting) studies, CTs or MRIs. They relied only upon their clinical skills and acumen. As time and medical technology have evolved, additional studies have corroborated their clinical suspicions, lending further credence to their clinical acumen.

While it can safely be said that the medical community, and society in general, did not recognize child abuse as a valid entity until the mid-twentieth century, it was a French forensic physician, Auguste Ambroise Tardieu (fig. 1), who penned the first detailed medical description of child abuse in his 1860 publication Etude Medico-Legale sur les Sevices et Mauvais Traitements Exerces sur des Enfants (Forensic Study on Cruelty and Ill Treatment of Children; fig. 2). [113] Tardieu was the leading forensic expert of his time, holding prestigious positions such as dean of the faculty of medicine at the University of Paris and president of the French Academy of Medicine. [114] He published works on child physical abuse, child sexual abuse, and child labor laws. [115]

In his 1860 publication, Tardieu detailed thirty-two cases of child abuse, describing bruises of varying colors, skeletal fractures, and subdural hemorrhages (SDHs). [116] Tardieu also described findings of infanticide, including cases without external signs of injury, but where hemorrhage in the brain and collections of blood *524 over the brain were described. [117] In these writings, Tardieu clearly expressed his belief that the abuse was inflicted by parents or caretakers of the child. [118] Although his considerable influence led to revision of French child labor laws, Tardieu's works on child abuse went unappreciated and essentially ignored. [119]

The mid-to-late nineteenth century was a period of significant medical advancements. [120] Secondary to the works of Louis Pasteur and others, Germ theory became the predominant explanation for previously unexplained maladies. [121] Diseases such as scurvy, rickets, and even SDHs, were thought to be infectious. [122] A highly prominent physician, Rudolf Virchow, proposed the theory that SDHs, because they frequently presented with a membrane, were caused by inflammation and infection. [123] He

termed this theory "pachymeningitis hemorrhagica interna". [124] Because of Virchow's significant stature within the medical community, and because the theory fit within the greater framework of the prevailing germ theory, the inflammation/infection theory of SDHs ("pachymeningitis hemorrhagica interna") was accepted for many decades. [125]

It was not until the early twentieth century that trauma began to be realized as an important cause of SDHs. [126] While earlier reports of the 20th century (despite a significant lack of evidence) still tended to support infectious or nutritional deficits as the cause of the SDHs, ***525** later reports began to identify trauma as the primary etiology. [127] Additionally, many of those reports documented the association of SDHs, ophthalmic hemorrhages, and sometimes bone lesions in infants. [128]

In 1914, the prominent British neurosurgeon, Wilfred Trotter (fig. 3), published a report declaring trauma as the true cause of SDHs. [129] Trotter was a distinguished and accomplished physician who held many significant positions, not the least of which was his position as private physician to King George V. [130] Frustrated by the term "pachymeningitis hemorrhagica interna," Trotter asserted that the term presumed an infectious or inflammatory etiology and thus was a misleading hypothesis. [131] Trotter stated, "[h]aemorrhagic pachymeningitis is almost if not quite invariably a true traumatic haemorrhage coming from veins torn in their course between the brain and a dural sinus." [132] Trotter's work paved the way for other physicians, especially neurosurgeons, to re-examine the pathophysiology of SDHs. [133] As a consequence, multiple case reports by well-reputed physicians began to question other previously well-recognized causes-- syphilis, [134] hydrocephalus, [135] nutritional (scurvy), [136] and other infectious [137] --as the primary ***526** etiology for SDHs. [138]

Then, in 1946, Dr. John Caffey (considered by many to be the father of pediatric radiology), examined the correlation of SDHs and long bone fractures in a separate field of medicine--radiology. [139] After seeing repetitive cases of injuries over many years, Caffey published a case series of six infants with SDHs and long bone fractures. [140] In none of the six cases was there a historical report of trauma or of systemic disease. [141] Nevertheless, after systematically ruling out all other causes, Caffey concluded that trauma was the most logical etiology for these radiologic findings. [142] Caffey even associated the retinal hemorrhages in several of these cases to trauma. [143] Caffey, however, was reluctant to conclude inflicted injury in these cases. [144]

Secondary to Caffey's work, in 1953, another prominent radiologist, Frederic Silverman, catalogued radiographic signs of what he termed to be the "most common bone 'disease' of infancy": skeletal trauma. [145] In identifying trauma as the most common cause of SDHs and bone fractures in infants, Silverman meticulously ruled out all nutritional and metabolic causes. [146] In the two decades following Caffey's historic article, multiple articles from national and international authors confirmed the association of SDHs with ***527** inflicted trauma. [147]

It was not until 1962 that the work of an eminent pediatrician, C. Henry Kempe (fig. 4) and his colleagues (radiologist Frederic Silverman and psychiatrist Brandt Steele) brought the issue of child abuse to the medical and national forefront. In their landmark article, The Battered-Child Syndrome (fig. 5), Kempe et al. carefully and thoughtfully described a syndrome of various injuries, including SDHs, that resulted from trauma. [148] However, unlike the vast majority of physicians that preceded them, Kempe et al. concluded that these injuries resulted from the intentional acts of parents or other care-givers. [149] Kempe et al. stated that abuse:

> should be considered in any child exhibiting evidence of fracture of any bone, subdural hematoma, failure to thrive, soft tissue swellings or skin bruising, in any child who dies suddenly, or where the degree and type of injury is at variance with the history given regarding the occurrence of trauma. [150]

In support of their conclusions, the authors had surveyed 71 hospitals nationwide, with a report of over 300 cases in which 33 children had died and 85 had suffered permanent brain damage in one year. [151]

***528** As a consequence of Kempe et al.'s historic work and the general medical community's increasing acceptance of child abuse as a viable medical diagnosis, case reports continued to publish the presence of concurrent SDHs, retinal hemorrhages, and bony lesions in infants, often without external signs of trauma. [152] Finally, in the early 1970s, based upon the work of Wilfred Trotter, numerous case reports, and the experimental biomechanical evidence of Ommaya and his colleagues, [153] a British neurosurgeon, A. Norman Guthkelch, and the father of pediatric radiology, John Caffey, proposed shaking or whiplash injury as the cause of infantile SDHs. [154]

In theorizing that multiple acceleration and deceleration events, caused by head shaking, resulted in the intracranial injuries, Guthkelch stated that, "the relatively large head and puny neck muscles of the infant must render it particularly vulnerable to whiplash injury." [155] Meanwhile, Caffey published a series of case reports identifying the "pattern of concurrent SDHs, [sometimes] bony lesions, and retinal hemorrhages in infants thought to be injured by shaking." [156] In fact, in the words of two learned authors: "It is difficult to comprehend how the common association between SDH and skeletal injuries, and the etiologic factors [trauma] linking the two, could have eluded the scrutiny of all but a handful of physicians and surgeons dealing with children until Caffey reported ***529** his historic observations." [157]

B. "A Flawed Science"? [158]

As mentioned earlier, certain legal scholars have asserted that "the scientific underpinnings of SBS have crumbled over the past decade," [159] that the medical research underlying "SBS is a flawed science" [160] predicated upon "circular reasoning," "data gaps," and "inconsistency of case definition," [161] and that "as technology and scientific methodology advanced, researchers questioning the basis for SBS reached a critical mass." [162] In order to appropriately assess the sufficiency of the scientific evidence underlying AHT/SBS, some basic statistical concepts must be discussed.

1. Basic Statistical Principles & Quality of Evidence

Statistical evidence is an important complement to the practice of clinical medicine. Statistical evidence can offer probabilities and estimations of the risk of disease states in certain patient populations. It can help guide determinations of appropriate and inappropriate diagnostic testing in certain clinical scenarios. Moreover, it can provide empirical support for optimal therapeutic interventions in cases where treatment is warranted. However, statistical evidence cannot substitute for clinical judgment. It is a complement, not a replacement.

The field of statistics generally encompasses collecting, analyzing, presenting, and drawing inferences from data. [163] For the limited purposes of this article, we will review the general statistical ***530** principles involved in collecting and drawing inferences from data. [164]

a. Collection of Data

It has been stated that "[a]n analysis is only as good as the data on which it rests." [165] The attainment of valid, reliable data is, to a large extent, determined by the design of the study. [166] When the issue is causation, there are three general types of explanatory information provided: anecdotal evidence, observational studies, and controlled experiments. [167] Each of these types of information has its limitations. [168]

Anecdotal reports, while offering information that can be the stimulus for further study, can be misleading and, therefore, are insufficient to conclusively establish association. [169] Observational studies can provide strong evidence of association, but further analysis is necessary "to bridge the gap from association to causation." [170] And controlled experiments, while ideal for determining causation, are often too expensive and cumbersome to undertake. [171] Examples of observational studies include case reports or case reviews, where as examples of controlled experiments include randomized controlled trials (RCTs) or non-randomized ***531** controlled studies (such as non-randomized dose finding studies). [172] Because observational studies and controlled experiments are the more reliable types of information, [173] it is important to understand the value of, and distinction between, the two.

"In a controlled experiment, the investigators decide which subjects are exposed to the factor of interest and which subjects go into a control group." [174] In "observational studies, the subjects themselves choose their exposures." [175] Thus, in observational studies, the experimental" (or "treatment") group will most likely differ from the control group "with respect to . . . [many] factors other than the one of primary interest. [176] These many "other factors" are also known as "confounding variables," and could be limitations to the validity of the results if not properly accounted for in the design of the study. [177] "In randomized controlled experiments, investigators assign subjects to [experimental (or "treatment") and] control groups at random." [178] By assigning subjects randomly to either the experimental or control groups, the investigator "tends to balance the groups with respect to possible confounders," thus enhancing the likelihood that the groups are comparable except for the factor of interest (or treatment). [179]

It is noteworthy that "[t]he bulk of the statistical studies . . . [presented] in court are observational, not experimental." [180] Observational studies (i.e., case reports and case reviews) can provide compelling evidence when certain circumstances are present:

***532** 1) When "[t]he association is seen in studies of different types among different groups" ("This reduces the chance that the observed association is due to a defect in one type of study or a peculiarity in one group of subjects.");

2) "[W]hen the effects of plausible confounding variables are taken into account by appropriate statistical techniques;" and

3) When "[t]here is a plausible explanation for the effect of the independent variables." [181]

In general, "observational studies succeed to the extent that their [experimental (or treatment)] and control groups are comparable." [182] If a study is well designed, accounting for confounding variables, it is deemed to be internally valid. [183] However, the generalization of the conclusions of a study, or its "external validity," is a different matter. [184] Finally, a study is "reliable" if its results are reproducible by scientists in separate studies. [185]

In the realm of clinical medicine, observational studies are not just the norm but the cornerstone of medical diagnoses. Almost all well-established, undisputed medical diagnoses have no randomized controlled trials (RCTs) supporting or validating their diagnostic criteria. For example, migraine headaches have an extensive historical basis in the medical literature for evaluation, diagnosis, and therapy. In fact, the International Headache Society lists clear diagnostic criteria for migraine headaches, and provides the most up-to-date medical literature in support of that diagnostic criterion. [186] Yet, throughout the extensive body of medical literature on migraine headaches, there is not one RCT evaluating the diagnostic criteria for migraine headaches, or their validity. But ***533** there is no dispute regarding the validity of migraine headaches as a medical diagnosis. [187] Such is also the case for multiple other well-established, undisputed, common medical diagnoses--viral upper respiratory infections (the common cold), community acquired pneumonia, otitis media (ear infection), depression, and all other psychiatric disorders. In

short, the requirement that an RCT is necessary in order to validate diagnostic criteria of a particular medical diagnosis is not only inaccurate but also inconsistent with the vast majority of clinical medicine. [188]

At this point, it is relevant, and important, to examine one piece of medical literature which is often cited by opponents [189] of AHT as evidence of the paucity of sound medical literature on AHT (SBS): "Evidence-Based Medicine and Shaken Baby Syndrome Part 1: Literature Review, 1966-1998." [190] In this four-page article, the author proffers "neutrality," and proceeds to educate the reader about properly conducted studies, with sound methodological design, which fall into a "quality of evidence ratings" system. [191] Based upon the author's search of the Medline database, and the Internet via "Internet Explorer," using only the search term "shaken baby syndrome," the author finds only seventy-one articles (in a span of thirty-two years of medical literature) on the topic of AHT (SBS). [192] The author then reduces those seventy-one articles to fifty-four because some of the articles "only peripherally mention" SBS or are somehow "unrelated" to SBS. [193] Of those fifty-four remaining ***534** articles, the author finds only one "randomized control trial" and twenty-six case series (twenty-five retrospective and one prospective), and a total of 307 cases of SBS. [194] Based upon the author's review of this literature, he concludes that in studies conducted before 1999 there exist "serious data gaps, flaws of logic, [and] inconsistency of case definition" in SBS; catch-phrases which have been frequently reified in some medical and legal literature. [195] Consequently, the author concludes that "the commonly held opinion that the finding of SDH and RH in an infant was strong evidence of SBS was unsustainable, at least from the medical literature." [196]

Evidence-Based Medicine and Shaken Baby Syndrome Part 1: Literature Review, 1966-1998 is a prime example of poor medical literature, which somehow makes its way into a medical publication. Ironically, the article itself suffers from fatal methodological flaws and data gaps, but professes to assess the methodology of SBS studies and finds "data gaps" in them. [197] It is unclear why, and unacceptable that, the author chooses to conduct his search with the confining search term of "shaken baby syndrome." The author fails to search other common terms such as "inflicted neurotrauma," "non-accidental trauma," "whiplash shaken infant/baby syndrome," or even more general terminology such as "subdural hemorrhage/hematoma" or "retinal hemorrhage." [198] Because of this methodological flaw, as will be demonstrated below, the author misses the vast majority of literature on AHT and even the seminal articles by Guthkelch and Caffey. [199] Additionally, the author offers no critical analysis of any of the articles cited, no assessment of the designs of any of the individual studies, no reference to the statistical information, and no analysis of any of the statistical data ***535** or the inferences drawn from them. [200]

Finally, the author incorrectly uses the quality of evidence ratings system. The author asserts that the best evidence is "Level 1" quality of evidence (RCTs), and this is not found in the diagnostic studies involving AHT/SBS. [201] However, as discussed above, RCTs (the "Level 1" quality of evidence) are NOT appropriate for diagnostic studies. The AHT literature, like many other diagnoses (such as migraine headaches), should not be criticized for the existence of a "higher" level of evidence that is inappropriate to the question being asked. Thus, even the most ardent EBM advocate would admit that the best quality of evidence that can be expected in diagnostic studies is "Level 2" evidence (well-designed case series). And of this, as will be detailed below, there is abundant evidence in the AHT literature.

It is troubling that legal scholars and some courts have relied upon this article as an adequate assessment of the medical literature surrounding AHT. [202] Any future reliance upon this article should be seriously questioned.

b. Drawing Inferences from Data

Upon attainment of data, an investigator must determine what significance should be given to that data. In so doing, the investigator must determine whether the results obtained are attributable to random error. [203] Did "chance" produce the results? [204] Would a different pattern emerge if more data were collected? [205] In assessing the potential impact of chance error,

an investigator must consider the precision of the data (i.e., the standard deviation and *536 degree of confidence) and the statistical significance (the p-value) of the data.[206]

In assessing precision of the data, a standard deviation (or standard error) gives the investigator an estimate of the magnitude of random error.[207] A standard deviation is a variability range of data from the "mean" of the data.[208] Assuming a normal distribution of data, one standard deviation from the mean of data is commonly understood to encompass 68% of the data.[209] For example, the average height for adult women in the United States is about 64 inches, with a standard deviation of around 3 inches.[210] This means that most women (about 68%, assuming a normal distribution) have a height within 3 inches of the mean (61-67 inches).[211] Two standard deviations from the mean encompass 95% of the data.[212] Thus, in our example with height of adult women in the United States, two standard deviations would be a height within 6 inches of the mean, or 58-70 inches. Since the standard deviation "measures the likely size of the random error[, i]f the standard deviation or error is small, the estimate probably is close to the truth."[213]

Confidence intervals are another manner of expressing reliability in the interval data.[214] Again, assuming a normal distribution curve, a 95% confidence interval indicates a range of data from -2 standard deviations to +2 standard deviations.[215] "A *537 high confidence level alone means very little, but a high confidence level for a small [data] interval is impressive, indicating that the random error in the . . . [interval] is low.[216]

In assessing statistical significance, it is important to understand the concept of the p-value. The p-value is "[t]he probability of getting, just by chance, a test statistic as large as or larger than the observed value."[217] In more simple terms, it is the probability the result obtained is secondary to chance.[218] In social sciences and medicine, this "observed significance level" (the p-value) is usually set at 5% (or 0.05) for "statistically significant," 1% (or 0.01) for "moderately high" statistical significance, and 0.1% (or 0.001) for "high or strong" statistical significance.[219] Thus, "[i]f p is smaller than 5% [(or 0.05)], the result is said to be 'statistically significant.'"[220] Small p-values speak against the hypothesis that the *538 result can be explained by chance, while large p-values indicate that chance cannot be ruled out as an explanation for the data.[221]

A few other statistical concepts in clinical medicine are important to discuss briefly: "sensitivity," "specificity," "positive predictive value," "negative predictive value," and "odds ratio." "Sensitivity" is "the probability that a test for a disease will give a positive result" when the patient actually has the disease.[222] Put simply, it is actually the chance the condition will be found by the test.[223] "Specificity" is "the probability that a test for disease will give a negative result when the patient does not have the disease."[224] Put simply, it is the chance that someone without the disease will actually have a negative test.[225] "Positive predictive value" is the proportion of patients who have positive test results and actually have the disease or condition.[226] This value is very important in diagnostic testing as it reflects the probability that a positive test reflects the underlying condition being tested.[227] "Negative predictive value" is the "proportion of patients with negative test results who are correctly diagnosed."[228] "An "odds ratio" is a way of comparing whether the probability of a certain event is the same for two groups."[229] "An odds ratio of one implies that the event is equally likely in both groups.[230] An odds ratio greater than one *539 implies that the event is more likely in the first group."[231]

2. The Statistical Evidence

The peer-reviewed medical literature on the topic of AHT is voluminous. It is somewhat confusing how any author could assert there is a paucity of "quality" medical literature on the topic.[232] In hopes of clarifying and substantiating this matter, this author has compiled a brief bibliography (Appendix A)[233] of the peer-reviewed medical literature on the topic, organized by types

of articles in the various subspecialties, so the reader may judge the literature for himself/herself.[234] A critical analysis of the quality of some of that literature will be discussed herein below.

In general, there have been at least two treatises, comprising more than 880 pages, on the topic of AHT.[235] Additionally, there are at least 14 chapters, comprising another approximate 260 pages, on the topic of AHT within larger child maltreatment/ abuse texts.[236] In addition to that, there are over 700 peer-reviewed, clinical medical ***540** articles,[237] comprising thousands of pages of medical literature, published by over 1000 different medical authors, from at least 28 different countries[238] on the topic of AHT. Furthermore, the topic of AHT has been examined, studied, and published in the following disciplines: biomechanical engineering, general pediatrics, neonatology, neurology, neurosurgery, nursing, obstetrics, ophthalmology, orthopedics, pathology (including forensic pathology), radiology, and rehabilitative medicine.

With regards to the "quality" of medical literature, it bears remembering that retrospective reviews are not inherently (by the nature of being retrospective or non-randomized) unreliable. It is the design of the review and the quality of the analysis (i.e., accountability for bias, confounding variables, interpretation of data, etc.) that determines the validity of the results. Nevertheless, even with that proviso, there have been at least eight systematic reviews, over fifteen controlled trials, over fifty comparative cohort studies or prospective case series, and numerous well-designed, retrospective case series/reports, comprising thousands of cases, supporting the diagnosis of AHT.[239] As will be discussed in detail below, in this author's review of all of the published, peer-reviewed, clinical ***541** medical literature (greater than 700 articles), there is not one clinical study that demonstrates a greater statistical association of either subdural hemorrhages or retinal hemorrhages with accidental trauma over abusive head trauma. Additionally, since there has been criticism of the questionable "quality" of the medical literature supporting AHT (i.e., a lack of randomized, controlled studies),[240] it is important to note that almost all of the papers "questioning" the validity of AHT (save two or three) are non-randomized, retrospective case series/reports, and without comparative control groups. In fact, many are single case reports.

a. Subdural Hemorrhages

The differential diagnosis (i.e., list of potential causes) for subdural hemorrhages (SDHs) is extensive. A summarized list of those causes is detailed in Appendix B. When traumatic, the mechanism for the SDH is either a contact (or impact) force or an inertial (acceleration-deceleration) force or both.[241] "Contact . . . [forces] cause damage at the site . . . [where] contact occurs.[242] "Disruption of the skull's integrity secondary to the contact force can result in a disruption of the underlying blood vessels and consequent development of a hemorrhage.[243] These hemorrhages can be epidural (outside the dura mater), subdural (in the potential space underneath the dura mater), or, sometimes, intradural (within the layers of the dura).[244] In inertial events, the acceleration-deceleration motion of the brain results in strain upon the cortical bridging veins, which exceeds their tolerance levels and subsequently leads to rupture and hemorrhage (subdural and/or subarachnoid).[245]

Although there are many potential causes of SDHs, several ***542** studies indicate that trauma is the most common cause.[246] In one such prospective study of all infants ages zero to two in the U.K. and the Republic of Ireland, from 1998 to 1999, Hobbs et al. identified 186 infants with SDHs (by CT, MRI, ultrasound, or post-mortem examination).[247] Of the 186 infants with SDHs, 113 (61%) had SDHs caused by trauma, 30 (16%) by infection or other non-traumatic medical cause, and 43 (23%) by an undetermined cause.[248] Of the 113 traumatic SDHs, 106 (94%) were determined to be of non-accidental etiology, and only 7 (6%) were determined to be accidental.[249] Similar results were noted in retrospective reviews by Jayawant et al. in Wales and southwest England from 1993 to 1995, Trenchs et al. in Barcelona, Spain from 1995 to 2005, and Tzioumi & Oates' in the Royal Alexandra Hospital for Children in Australia.[250]

Feldman et al. confirmed a predominance of non-accidental injury over accidental injury as the etiology of SDHs in their 2001 prospective study of 66 children, under age three, with SDHs.[251] Feldman et al. excluded patients that presented with SDHs secondary to known hemorrhagic disease (i.e., bleeding disorder), prior neurosurgical procedure, previously recognized perinatal (i.e., near birth) brain injury, or infection.[252] In efforts to avoid "circularity" concerns, Feldman et al. designed their study such that retinal hemorrhages (RHs) were not a part of the classification *543 criteria for intentional injury.[253] In their cohort, Feldman et al. found that of the 66 patients, 39 (59%) patients were confirmed as suffering intentional injury, 15 (23%) were unintentional or accidental, and 12 (18%) were undetermined.[254]

Pathology studies have also confirmed the predominance of trauma, and more specifically non-accidental trauma, as the cause of SDHs.[255] In 2009, Matschke et al., published the results of their fifty-year retrospective review of the causes of death for infants less than one year old.[256] Of 715 infant deaths, only 50 infants (7%) were identified with SDHs.[257] Of those 50 SDHs, 15 (30%) were traumatic, 13 (26%) were secondary to bleeding/clotting disorders, 13 (26%) were perinatal, 4 (8%) were infectious, 4 (8%) were undetermined, and 1 (2%) was secondary to metabolic disease.[258] Of the traumatic SDHs, 14 (93%) were secondary to non-accidental trauma, and only 1 (7%) was accidental.[259] Thus, Matschke et al. concluded that "most . . . [SDHs are] attributable to trauma, with NAHI [(Non-Accidental Head Injuries)] substantially outnumbering accidental injuries . . . ."[260]

Although SDHs are not specific[261] for non-accidental injury, several well-designed prospective studies demonstrate a significant and strong association of SDHs with non-accidental/inflicted trauma over accidental trauma.[262] In 1992, Duhaime et al. published *544 the results of their prospective study of 100 patients less than two years of age who suffered head injuries.[263] In efforts to avoid "circularity" concerns, Duhaime et al. used strict criteria for determining "inflicted" injury.[264] The authors excluded retinal hemorrhages (RHs) as a diagnostic criterion, and they only included SDHs that had no history of trauma but had clinical or radiologic findings of blunt impact to the head.[265] Thus, the authors designed an algorithm, which was "deliberately biased to reduce false positives and thus may underestimate the true incidence of child abuse."[266] In Duhaime et al.'s cohort, 76 patients were determined to be from accidental causes and 24 were determined to be "inflicted."[267] Duhaime et al. found that only 3 out of 76 (8%) patients in the accidental group had SDHs, while 13 out of 24 (54%) patients in the "inflicted" group had SDHs.[268] This computed to a p-value of less than 0.0002, meaning these findings could have occurred by random chance no more than two times in 10,000 patients.[269] Thus, Duhaime et al. concluded that the relationship between inflicted injury and SDHs was highly statistically significant.[270]

In 2004, Bechtel et al. produced similar results.[271] The authors prospectively studied 82 children, age zero to twenty-four months, who were admitted to Yale New Haven Children's Hospital from *545 August 2000 to October 2002 for head trauma.[272] In avoiding "circularity" concerns, the authors classified "inflicted" head injury only if there was clear evidence of head injury and no trauma history provided, if there was a traumatic history incompatible with the developmental capabilities of the infant, if there was a confession of inflicting the injury, if there was a witnessed inflicted injury, or if there was evidence of other physical injuries which were characteristic of inflicted injury (e.g., patterned bruises, etc.).[273] The authors did not include RHs in the diagnostic criterion of "inflicted" injury.[274] Of the eighty-two patients, sixty-seven were determined to be "accidental," and fifteen were determined to be "inflicted."[275] Bechtel et al. found that 12/15 (80%) patients in the "inflicted" head injury group had SDHs, while only 18/67 (27%) patients in the "accidental" head injury group had SDHs.[276] This computed to a p-value of less than 0.001.[277] Again, this meant that these findings could have occurred by chance or randomly no more than one in 1,000.[278] Thus, Bechtel et al. also concluded that the association of SDHs with inflicted injury was highly statistically significant.[279]

In 2010, Vinchon et al. published the results of their prospective series of eighty-four patients, from 2001 to 2009, with independent corroboration of head injury.[280] In Vinchon et al.'s cohort, thirty-nine patients were witnessed accidents and forty-five patients were confessed inflicted head injury.[281] Only 17 out of 39 (44%) witnessed accidents had SDHs, while 37 out of 45 (82%) inflicted head injury ***546** patients had SDHs.[282] This computed to a p-value of less than 0.001.[283] As with Duhaime et al. and Bechtel et al., Vinchon et al. concluded that the association SDHs with non-accidental injury was highly statistically significant.[284] Several other well-designed, prospective and retrospective general pediatric studies have found similar results and come to the same conclusion.[285]

Radiology studies have helped to further characterize the appearance of SDHs seen in AHT cases. Multifocal SDHs, interhemispheric SDHs (located between the two hemispheres of the brain), and convexity SDHs (located at the front or back "curves" of the brain) have a stronger statistical association with non-accidental trauma than with accidental trauma.[286] In 2002, Wells et al. published the results of a retrospective review of the CTs of 293 children, under age three, with intracranial hemorrhage at the ***547** Children's Hospital of Wisconsin from 1991 to 2001.[287] Blinded to the CT findings, an injury was classified as "intentional if there was a confession of abuse, the injuries were incompatible with the stated mechanism of injury, or the caretaker offered no explanation for the injuries."[288] "An injury was classified as "unintentional if it was witnessed by someone other than the caretaker or there were no discrepancies between the described mechanism and the physical findings."[289] Then, blinded to the clinical findings, a pediatric radiologist reviewed the CT findings for the presence and location of intracranial hemorrhage and other intracranial abnormalities.[290] Wells et al. found that 105 out of 148 (71%) intentional injury patients had an interhemispheric SDH, while only 21 out of 109 (19%) unintentional injury patients had an interhemispheric SDH; and, 99 out of 148 (67%) intentional injury patients had a convexity SDH, as compared with 14 out of 109 (13%) unintentional injury patients.[291] For both these injuries, this computed a p-value of less than 0.05.[292] Thus, Wells et al. concluded that there was a statistically significant association with convexity and interhemispheric SDHs and intentional injury.[293] Similar results were produced by Hymel et al. and by Datta et al.[294] Additionally, in the Datta et al. study, there was a statistically significant association with multifocal SDHs and non-accidental injury.[295]

***548** Thus, with regards to the validity and reliability of the statistical evidence on SDHs and AHT, there are several well-designed prospective studies and retrospective reviews. Additionally compelling is that the statistical results are similar along multiple lines of research-- pathology, radiology and general pediatrics. All have produced the same results: the significant statistical association of SDHs with non-accidental trauma over accidental trauma. This author's review of the evidence-based medical literature has revealed no published, peer-reviewed clinical studies that conclude differently.

b. Retinal Hemorrhages

The retina is the multi-layered, inner lining of the eye.[296] The posterior pole is the area of the retina that encompasses the major blood vessels, the macula, the fovea, and the optic nerve head (the optic disc).[297] The fovea is the area of the retina where the central visual axis through the pupil falls.[298] The area of retina surrounding the fovea is the macula.[299] These structures are depicted in Figures 6 and 7.

In young children/infants, the vitreous gel that fills the eye is adhered much more strongly to the macula, peripheral retina, and the retinal blood vessels as they course on the retinal surface.[300] This difference in anatomy from the adult eye is relevant to the theory of how RHs are formed (repetitive acceleration-deceleration forces) in the setting of AHT.[301]

"Hemorrhages [can] occur on the surface of the retina (preretinal), under the retina (subretinal), or within the retinal ***549** [layers (intraretinal)]."[302] Hemorrhages can have a certain appearance and size, and can be confined to the posterior pole or

extend to the ora serrata (the edges of the retina).[303] "Flame" or "splinter" RHs are hemorrhages that lay in the superficial nerve fiber layer of the retina.[304] "Dot" and "blot" RHs are round and amorphous-shaped hemorrhages within the deeper layers of the retina.[305] An important form of RHs is retinoschisis--where there is splitting of the retinal layers with blood accumulating in the intervening space.[306] Retinoschisis can sometimes be accompanied by circumlinear pleats or folds in the retina at the edges of the schisis.[307] Retinoschisis with pleats or folds is an important finding, because, other than AHT, in children younger than five years it has only been reported in two cases of fatal crush injuries to the head, one case of leukemia, and in cases of severe, fatal motor vehicle accidents.[308]

Mild RHs are generally understood to be a few, dot/blot or flame/splinter-shaped RHs, in the intraretinal or preretinal layers, and confined to the posterior pole.[309] Severe RHs are generally understood to be diffuse, too numerous to count hemorrhages, extending to the periphery of the retina (not confined to the ***550** posterior pole), usually involving multiple layers of the retina (intraretinal, preretinal or subretinal), and sometimes accompanied by retinoschisis with or without folds.[310] Mild RHs, severe RHs, and retinoschisis are depicted below in figures 8, 9, 10.

As with SDHs, the differential diagnosis for subdural hemorrhage RHs is extensive. A summarized list of those causes is detailed in Appendix C. Assessing the diagnostic significance of RHs requires the consideration of other medical causes and an understanding of the spectrum of injury patterns observed in accidental trauma. Through the inferential and deductive process of eliminating other potential mechanisms, one recognizes the significant probability that repetitive acceleration-deceleration forces are the causative mechanism of severe RHs.[311]

While several studies demonstrate an association of RHs with birth, several factors distinguish birth-related RHs from the RHs commonly seen in AHT.[312] First, the vast majority of birth-related retinal hemorrhages are intraretinal.[313] Multi-layered RHs, as commonly seen in AHT, have not been reported in the medical literature in association with birth.[314] Second, study of the natural history of birth-related RHs reveals that the vast majority of these RHs resolve by two to four weeks of life.[315] This led one author to conclude that RHs "in infants older than 1 month . . . [are] not likely related to birth".[316] Finally, retinoschisis (splitting of the retina) has ***551** never been reported in association with birth injury.[317]

The commonality, and somewhat similarity, of birth-related RHs and the RHs commonly seen in AHT compels one to consider increased intracranial pressure or increased intrathoracic pressure as potential causative mechanisms for RHs.[318] Additionally, because rib fractures are occasional concurrent injuries in AHT cases, increased intrathoracic pressure is naturally thought to be implicated.[319]

Studies examining the effects of chest compressions in CPR (cardio-pulmonary resuscitation) have failed to demonstrate any severe RHs (the kind seen in AHT).[320] In one such study, Odom et al. prospectively examined the prevalence and character of RHs in patients in a pediatric ICU who had received at least one minute of chest compressions and survived.[321] After excluding patients that had evidence of trauma, documented retinal hemorrhages before CPR, suspicion of child abuse, or diagnosis of near-drowning or seizures, Odom et al. found 43 patients that met inclusion criteria.[322] In fact, "[a]ll of the precipitating events leading to cardiopulmonary arrest occurred in their intensive care unit, eliminating the possibility of physical abuse as an etiology."[323] Of the 43 patients, "[t]he mean duration of chest compressions was 16.4 minutes . . . with 58% lasting between 1 and 10 minutes. Five patients had chest compressions lasting less than 40 minutes, and two patients had open chest cardiac massage. All patients survived their resuscitative ***552** efforts."[324] Odom et al. found small punctate retinal hemorrhages in only one patient.[325] There was no patient with severe RHs.[326] Well-designed studies involving other clinical scenarios that increase intrathoracic pressure, e.g., coughing, vomiting, or seizures, also have failed to demonstrate any of the type of severe RHs commonly seen in AHT.[327]

With regards to increased intracranial pressure as a cause for severe RHs in children, in 2002, Schloff et al. published the results of a prospective study, which was designed to find the incidence of RHs in children with intracranial hemorrhage and increased intracranial pressure (also known as Terson's syndrome).[328] Only children from known non-abuse cases were included in their study.[329] Of the 57 children studied, 27 were from known accidental trauma (MVA's, sports accidents, falls, etc.), 24 from surgeries, and six from other causes (vessel malformations, infection, etc.).[330] Fifty- *553 five out of fifty-seven children (96%) had no evidence of RH.[331] "One . . . [child] had a single dot hemorrhage associated with [a] presumed infectio[n] . . . . The second . . . [child] had three flame and two deeper dot intraretinal hemorrhages."[332] She was the victim of a motor vehicle accident.[333] No child had severe or multi-layered RHs.[334] These results accord with the retrospective review conducted by Morad et al., also published in 2002.[335]

Furthermore, the postulated mechanism of RHs in the setting of increased intracranial pressure--obstruction of venous outflow from the eye (i.e., blood flowing out of the eye, through the head, and back towards the heart)--produces a pattern of hemorrhages that is not the pattern of hemorrhages seen in AHT.[336] The accidental head injury literature also demonstrates no severe RHs, and many of the children in those studies experienced increased intracranial pressure.[337]

On the other hand, several lines of research and analysis point towards acceleration-deceleration forces at the vitreo-retinal interface (remembering, from above, that the anatomy of an infant is *554 such that the vitreous gel is much more strongly adherent to the retina than in adults) as the causative mechanism for severe RHs.[338] First, "the pattern of hemorrhages . . . [in severe RHs] correlates with the . . . anatomy [of the eye in] the young child where[] the vitreous is most adherent with blood vessels" (in the periphery of the retina, and in the area of the posterior pole where retinoschisis occurs).[339] Second, severe RHs are not commonly seen in single acceleration-deceleration traumatic events (such as motor vehicle accidents and falls).[340] Third, in fatal cases, postmortem studies reveal that the vitreous is often still attached at the top of retinal folds, indicating a traction mechanism.[341] Finally, as will be detailed below, there is an extremely high, statistically significant association of severe RHs with AHT.[342]

In 2005, Vinchon et al. sought to study the diagnostic significance of RHs in cases of child abuse.[343] Their prospective study of 150 children included all children under two years old, who were admitted with head injury over a three year period.[344] Utilizing the strict algorithmic criteria of Duhaime et al. (discussed above) for determining "inflicted" injury, Vinchon et al. identified 57 cases of abuse, 88 eighty-eight accidental cases (household, birth trauma, and traffic accidents), and five undetermined.[345] Retinal data was available for 129 children (56 abuse, 73 accidents).[346] Moderate to severe RHs were found in 37 cases, all of them "abuse".[347] Vinchon et al. found the sensitivity, specificity, and positive predictive value of moderate or severe RHs for abuse to be 66.1%, 100%, and 100%, *555 respectively.[348]

Vinchon sought to re-examine this data, and its reproducibility, except this time with independent corroboration of head injury, so as to avoid any "circularity" concerns in his design.[349] In 2010, Vinchon et al. published the results of a prospective series of 84 patients who sustained injuries from either witnessed accidents (N=39) or confessed inflicted head injury (N=45; obtained from judicial sources).[350] Of the thirty-nine witnessed accidents, only one patient (2.5%) had moderate or severe RHs--that is the patient had a known impact to his head.[351] Of the 45 confessed inflicted injury patients, 34 (76%) had moderate or severe RHs.[352] Conversely, 34 out of 39 (87%) accident patients had mild or no RHs; and, 10 out of 45 (22%) of the inflicted head injury patients had mild or no RHs.[353] This data is graphically depicted (figure 11) below, and computed to a p-value of less than 0.001 (0.1%).[354] In further statistical analysis, Vinchon et al. determined the specificity and positive predictive value of severe RHs for abusive injury to be 97% and 96%, respectively.[355] Vinchon et al. calculated the specificity of SDH, RH

and the absence of evidence of impact to be 100% for abusive injury.[356] Thus, Vinchon et al. concluded that, in the absence of ocular impact, severe RHs were specific for inflicted head injury.[357] Similar results have been produced in well-designed prospective and retrospective studies by Pierre-Kahn et al., Bechtel et al., and Reece and Sege.[358]

***556** Pathology studies have produced similar results. Riffenburgh studied 197 confirmed child abuse deaths and compared them to 401 controlled patients (deaths secondary to auto accidents, drowning, SIDS).[359] Riffenburgh found 47% of child abuse deaths had RHs whereas only 4% of controls had RHs.[360] This computed to a p-value of less than 0.001 (0.1%), and an odds ratio of 18.9 for RHs and abuse.[361] Remembering "odds ratio" from the statistics section above, this means that RHs in abuse is almost nineteen times more likely than RHs in other circumstances (auto accidents, drowning, SIDs, etc).[362] Other authors have published comparable findings.[363]

In 2009, Maguire et al. published the results of their systematic review of all the scientific literature to identify clinical features that distinguished inflicted from non-inflicted brain injury.[364] After reviewing "20 [electronic] databases, websites, references and bibliographies, using over 100 keyword combinations," Maguire et al. identified over 6000 studies, which were relevant to the topic, and reviewed 320.[365] Secondary to strict inclusion criteria (including only those studies that compared the clinical features of inflicted and non-inflicted brain injury with consecutive case ascertainment), ***557** Maguire et al. found 14 studies that met those criteria, representing over 1600 children.[366] Cases were included only if strict definitional criteria for "inflicted" brain injury (i.e., those with witnessed abuse, confessions, legal decisions, or outcome confirmation by multi-agency child protection teams) was met.[367] The authors specifically excluded all studies where the decision of abuse relied solely on clinical features, so as to eliminate concerns for "selection bias" and "circularity."[368]

Conducting a multi-level logistic regression analysis, Maguire et al. found that RHs were "strongly associated with inflicted brain injury, with a positive predictive value of 71% and an odds ratio of 3.504."[369] Again, remembering odds ratios, based upon a comprehensive review of ALL the literature involving RHs, RHs are 3.5 times more likely to occur in inflicted circumstances than non-inflicted ones. The authors concluded, "By producing a multilevel logistic regression of specific clinical features on over 1600 children, we have shown that there is scientific evidence to support the distinction between [inflicted brain injury] and [non-inflicted brain injury] . . . . "This review is the largest of its kind, and offers for the first time a valid statistical probability of [inflicted brain injury] when certain key features are present (e.g., retinal haemorrhages)."[370]

In 2010, Bhardwaj et al. also published a systematic review of the diagnostic accuracy of RHs in AHT.[371] Upon examining three large medical databases, the authors identified 971 articles, and fifty-five met their relevance criteria for grading purposes.[372] Using a published grading checklist (designed to ensure the highest quality of design in studies), Bhardwaj et al. found twenty studies that met ***558** inclusion criteria.[373] Similar to the "Quality of Evidence Ratings system" employed by Donohoe (a ratings system that was used to critique the quality of literature behind AHT), Bhardwaj et al. found that the specificity of intra-ocular hemorrhages (RHs) for AHT was 94%.[374] The authors concluded:

> Currently, there is level II evidence from prospective controlled studies, supporting a significant relationship between IOH [(intraocular hemorrhage)] and AHT. . . . Level I evidence is impossible to achieve in this field, for obvious reasons. . . . Combined data from prospective studies of head injury indicate that IOH have a specificity of 94% for abuse.[375]

Thus, again, with regards to validity and reliability, there are two systematic reviews (comprising over thirty well-designed clinical studies and thousands of children), several well-designed prospective studies, and numerous retrospective reviews from multiple lines of research, general pediatrics, ophthalmology and pathology, all of which have produced the same results:

the highly significant statistical association of severe RHs with AHT. To this author's review of the evidence based medical literature, there are no published, peer-reviewed clinical studies that conclude differently.

c. Other Statistical Evidence

Well-designed comparative studies have demonstrated a statistically significant worse outcome (for both physical and cognitive functioning) for AHT patients over accidental trauma ***559** patients. In 1997, Haviland and Russell published the results of their comparative retrospective review of the outcomes of fifteen children, under age two, admitted to the pediatric ICU with AHT, and ten children, under age two, admitted to the same pediatric ICU during the same time-frame with known accidental head trauma.[376] Haviland and Russell followed the children for up to three years.[377] Of the AHT group, two patients died.[378] Of the remaining thirteen survivors, seven (54%) showed "severe" (meaning total mental and physical dependence) handicap, four (31%) had "moderate" (meaning partial paralysis, blindness, and developmental delay), one (8%) had "mild" (meaning partial paralysis and seizures), and only one (8%) was considered "normal" at a three-month follow-up.[379] Of the accidental group, one patient died.[380] Of the remaining nine survivors, only one (11%) had severe handicap, one (11%) had mild handicap, and seven were considered normal at discharge.[381] This computed to a p-value of less than 0.01 (1%).[382] Similar results were reproduced by Hymel et al., Vinchon et al., Keenan et al., and Ewing-Cobbs et al.[383]

***560** Other studies have focused on the significance of a discrepant clinical history to explain significant traumatic findings. A clear, biomechanically plausible account for how the injuries occurred should be available. When the history is absent, minimal, changing, or mechanistically implausible, suspicion of abusive injury is raised. In 2003 Hettler and Greenes, members of an emergency medicine group from Children's Hospital of Boston, examined the very issue of whether certain historical features are predictive of AHT.[384] Their retrospective review of 163 children, age three or younger, included patients admitted from 1993 to 2000 with acute traumatic intracranial injury.[385] The authors classified cases "as either 'definite abuse' or 'not definite abuse'. . . [based upon] radiologic, ophthalmologic, and physical examination findings, without regard to the presenting history."[386] Forty-nine out of 163 (30%) were classified as "definite abuse" and 114 out of 163 (70%) were classified as "not definite abuse."[387] Upon statistical analysis Hettler and Greenes found that no history of trauma had a 97% specificity and 92% positive predictive value for AHT.[388] When analyzed in the subgroup of patients with persistent neurologic abnormality at discharge, no history of trauma had a specificity of 100% and positive predictive value of 100% for AHT.[389] Studies by Duhaime et al. and Keenan et al. also confirm the association of discrepant clinical history and AHT.[390]

***561** d. Fallacy of Circular Reasoning, Alternative Hypotheses, & Data Gaps

i. "Circular Reasoning"?

It is appropriate at this point to address a criticism frequently levied against the medical literature on AHT: the logical fallacy of "circular reasoning."[391] While certainly some of the medical literature suffers from these design flaws, there are several factors not addressed by this critique. First, how does the logical fallacy of "circular reasoning," which essentially states a poor design of the medical studies, explain the associative findings of subdural hemorrhage and retinal hemorrhages found by Ingraham, Caffey, Guthkelch, Silverman, Kempe, and countless other historical authors, who reported these findings even before the diagnosis of Non-Accidental Injury existed?[392] What was their improper "design" in reporting these associative findings? Were these physicians somehow biased towards reporting these findings? Is it that these astute physicians were not rigorous or meticulous enough in their reasoning and evaluations to exclude other important causes such that the association of SDHs and RHs was not valid? Or is it that we are simply going to attribute the multiple reports of these associated findings to chance?[393] Why is it that multiple historical physicians, separated by significant geographical distance, in unrelated, various fields of medical study, and with no social or medical inclination to make these findings, collectively found the same associated

findings? In order to dismiss the associative strength of these findings (subdural hemorrhages and retinal hemorrhages), an appropriate response must first be given to all these historical physicians.

*562 Second, some circularity is inevitable, because we are unwilling to experimentally shake infants, and even reliably confessed accounts have some doubt. As detailed above, to the greatest extent possible, numerous well-designed studies set out to control circularity in their experimental design. When scientists critically examined those studies for bias secondary to circularity, not only was that bias lacking, but also scientists found results that were consistent with the rest of the clinical literature.[394] Therefore, although the possibility of circularity is present, and to some degree inevitable, we are unlikely to find substantially better evidence than we currently have for the absence of circularity.

Finally, telling evidence arguing against circularity is the absence of any large trials demonstrating a lack of association of either SDHs or RHs with AHT. If circularity were truly a valid criticism of the current clinical medical literature, in over twenty years of research on the topic, would there not exist one well-designed study that demonstrated a lack of association of either SDHs or RHs with AHT? Where is that study?

ii. Alternative Hypotheses

There have been two recent alternative hypotheses[395] for SDHs and RHs that have been the subject of some controversy--Geddes' "Unified Hypothesis," and Squier & Mack's "dural immature vascular plexus theory."[396] Geddes' Unified Hypothesis purports that "hypoxia [(lack of oxygen)], brain swelling and raised central *563 venous pressure cause blood to leak from intracranial veins into the subdural space, and that the cause of subdural bleeding in some cases of infant head injury is therefore not traumatic rupture of bridging veins, but a phenomenon of immaturity."[397] The essential components of this hypothesis are that hypoxic (lack of oxygen) injury to the brain results in increased intracranial pressure and brain swelling, which leads to "leaky" intracranial veins and subdural hemorrhage.[398]

The basis for Geddes' hypothesis was a cohort of fifty postmortem cases: seventeen fetuses, three spontaneous abortions, sixteen perinatal (within a week of life), five neonatal (within one month of life) and nine infant (within one year of life) deaths--all which resulted from non-traumatic causes.[399] Geddes et al. found microscopic intradural (within the layers of the dura, but not on the surface of the brain) blood in thirty-six of the fifty cases (72%).[400] However, if one excludes the fetuses and abortions, microscopic intradural blood was found in just thirteen of the thirty (43%) of the perinatal/neonatal/infant cases.[401] Macroscopic SDH (visible on the surface of the brain) was found in only one of the fifty cases (2%), an infant with overwhelming sepsis (infection).[402] Although an ophthalmologist was a co-author of the study, the authors did not examine or comment on retinal hemorrhages in their cohort.[403] Based upon the microscopic intradural findings, Geddes et al. hypothesized that intradural blood could "ooze" in the potential subdural space and result in macroscopic SDHs, although this did not occur in forty-nine out of fifty patients in their cohort.[404] Furthermore, based upon their data and calculations, Geddes et al. determined the p-value of hypoxia and macroscopic SDH to be *564 0.15.[405] Thus, based upon their own data, the authors could not even conclude that chance had been ruled out.[406] These results cannot be construed as statistically significant.[407] Geddes et al.'s results were three times higher than the highest limit of statistical acceptability (p=0.05).[408] This is truly notable when one compares it to the vast majority of statistical data supporting AHT (as discussed above), where p-levels are in the order of 0.01 to 0.001.[409]

Since the Unified Hypothesis was published in 2003, only one other peer-reviewed, clinical study has been published in the medical literature supporting this hypothesis.[410] In 2007, Cohen and Scheimberg published the pathologic results of a prospective series of twenty-five fetuses (age twenty-six to forty-weeks) and thirty neonates (age one hour to nineteen days) who suffered hypoxic (lack of oxygen)-ischemic (lack of blood) injury (HII).[411] Cohen and Scheimberg found macroscopic

SDHs in sixteen out of twenty-five (64%) fetuses, and twenty out of thirty (66%) neonates.[412] As with Geddes' study, no examination or comment was made on the concurrent presence or absence of RHs.[413]

The critiques of this study have been two-fold. One, it is well known that birth trauma is a cause of SDHs (secondary to dural tears involving the sinuses).[414] Thus, there was no explanation or ***565** accounting for this confounding variable.[415] It is not known how the authors distinguished which patients' SDHs were secondary to birth trauma and which were secondary to hypoxic-ischemic injury (HII).[416] Two, the age of Cohen and Scheimberg's patient cohort was not similar to the age of patients commonly involved in AHT.[417]

When considering Geddes' hypothesis that hypoxia (lack of oxygen) results in SDHs,[418] radiology studies are also helpful. Clinical radiology studies do not support an association of SDH and hypoxia.[419] MRI studies demonstrate that the pattern of hypoxic-ischemic injury (HII) in the brain is characteristically intraparenchymal (inside the brain tissue) hemorrhage, along with cortical (brain tissue) necrosis (death).[420] SDH is not a part of that pattern.[421] In 1998, Dubowitz et al. published the results of their retrospective review of the MRIs of twenty-two children (age six months to eleven years), who suffered HII (hypoxic-ischemic injury) after near drowning episodes.[422] While a variety of MRI findings were encountered, none of the patients had a SDH, and only one had a possible hemorrhage, and that was intraparenchymal.[423] Similar results have been published by Baenziger et al., Sie et al., Rutherford ***566** et al., and Barkovich et al.[424]

CT imaging has also failed to demonstrate SDHs in patients with HII.[425] In 2008, Rafaat et al. published their retrospective review of the CT findings in children suffering drowning episodes.[426] Of the 156 children included in their seventeen-year review, none had an intracranial hemorrhage.[427] Additionally, SDH is "conspicuously absent" from standard textbooks of neonatal neurology or MRI when addressing HII in infancy and childhood.[428]

Two recent pathology studies have evaluated the incidence of SDHs in HII cases. In 2007, Byard et al. published the results of a retrospective study of eighty-two fetuses, infants, and toddlers with proven HII and no trauma.[429] The cooperative study was undertaken by multiple forensic in Australia, the United Kingdom, Germany, Denmark, and the United States.[430] The age range of the eighty-two patients was thirty-five weeks gestation to three years.[431] All cases had histologically confirmed HII.[432] "Causes of the hypoxic episodes were . . . sudden infant death syndrome . . . [(SIDS)] (N = 30), drowning (N = 12), accidental asphyxia (N = 10), ***567** intrauterine/delivery asphyxia (N = 8), congenital disease (N = 6), aspiration of food/gastric contents (N = 4), inflicted asphyxia (N = 3), epilepsy (N = 1), dehydration (N = 1), drug toxicity (N = 1), complications of prematurity (N = 1), and complications of anesthesia (N = 1)."[433] In four instances, no initiating event was determined and "[i]n no case was there macroscopic evidence of subdural hemorrhage."[434]

In 2010, Hurley et al. published the results of a retrospective study of fifty children less than four years old who had suffered non-traumatic cardio-respiratory arrest and died at their institution between January 2001 and May 2007.[435] Specifically, the authors were looking to see whether there was a causal relationship between hypoxic-ischemic events (associated with cardio-respiratory arrest) and SDHs.[436] All children who had evidence of cranial trauma (even those with findings of occult head trauma on post-mortem examination) were excluded.[437] Additionally, other children were also excluded if they had evidence of a bleeding disorder, infection, metabolic, or degenerative neurological conditions.[438] The authors identified fifty children younger than four years of age who met their strict inclusion criteria; forty-eight of those fifty children were less than twenty-four months old.[439]

The average resuscitation time of children in the study was twenty-one minutes.[440] Forty of the fifty children died and had post-mortem examinations.[441] Thirty-nine of the forty post-mortem *568 examinations had no macroscopic evidence of SDH.[442] The one child (a 19-day old infant victim of an overlaying incident), who had macroscopic evidence of SDH, had a clot adhering to the dura, which the pathologist felt was consistent with birth-related trauma.[443] Of the five children in the study who had retinal examinations, none had RHs.[444] Thus, the authors concluded that "cardiopulmonary collapse per se and the attendant hypoxic-ischemic sequelae do not cause SDH."[445] The previously mentioned study by Matschke et al. identified similar results.[446]

The more recent version of Geddes' Unified Hypothesis is Squier and Mack's dural immature vascular plexus theory.[447] In this theory, the authors hypothesize that there is a plexus (network) of vessels within the dura mater that is immature and the most likely source for hemorrhage in non-traumatic conditions.[448] Akin to Geddes' Unified Hypothesis, the authors purport that hypoxia is the preeminent factor causing these immature vessels to leak, and subsequently result in SDHs.[449] However, also akin to Geddes' Unified Hypothesis, this theory offers no scientific data linking an intradural (within the dura) vascular plexus to the significant subdural hemorrhages in trauma.[450] Unlike even Geddes' Unified Hypothesis, this theory has not been studied in any cohort of patients. Thus, like Geddes' Unified Hypothesis, this theory is untested by the rigors of scientific falsifiability and unsupported by the medical literature. The legal analysis of these two hypotheses will be discussed in the Daubert analysis below.

*569 iii. Data Gaps

In every field of medicine, there are areas of incomplete information, where research and further investigation are beneficial. This is true for child abuse pediatrics as well. However, incomplete information does not necessarily equate to insufficient information. As previously outlined, vast amounts of historical reports, research data, and clinical experience have established quality, evidence-based information for the diagnosis of AHT with a reasonable degree of medical certainty.

That being said, some questions remain unanswered. Current areas in question include: 1) what are the exact tolerance and failure limits of the multiple intracranial structures (the dura mater, cortical bridging veins, the unmyelinated infant brain) of the human infant; 2) how do those structures, as well as other intracranial entities (such as cerebrospinal fluid), independently and collectively act to increase or decrease biomechanical forces; 3) what are the exact forces required to induce SDHs and DAI (Diffuse Axonal Injury) in the human infant brain; 4) what are the tolerance and failure limits of the infant cervical and thoracic spine;[451] 5) what is the exact mechanism for RHs and what role do multiple physiologic factors, such as increased intracranial pressure and biochemical (prostaglandins) release, play in their causation; and, 6) what are the incidence and prevalence of rare AHT "mimickers" (osteogenesis *570 imperfecta, glutaric aciduria type 1, etc.) in AHT cases. Ethical and logistical challenges may limit progress to research in the child abuse field.

These questions, and others, have already been identified by experts in the field of AHT as areas of present and future research.[452] Improvements in the biofidelity of anthropomorphic doll models, computer finite modeling of the intracranial and intraocular structures, and the identification of potentially specific biochemical markers of traumatic brain injury are just some of the examples of advancements in AHT research. Efforts to address these unknowns will only further enhance our understanding of AHT.

C. Coming to the Diagnosis of AHT

AHT is "those constellations of injuries that are caused by the directed application of force to an infant or young child, resulting in physical injury to the head and/or its contents."[453] Commonly observed injuries include scalp injury (e.g.,

bruises, lacerations/abrasions, swelling), skull fractures, intracranial (inside the skull) hemorrhage (i.e., SDH, subarachnoid hemorrhage, epidural hemorrhage, intraparenchymal hemorrhage), diffuse axonal injury,[454] cerebral edema (brain swelling), encephalopathy, cervical spine fractures, cervical spinal cord injury/hemorrhage, retinal hemorrhages, rib fractures, and long bone fractures. While any of the above injuries can result from, or accompany, AHT, the most common injuries associated with AHT are SDHs and RHs.

Recent legal literature and cases have cited a "diagnostic triad" of SDHs, RHs and encephalopathy as defining AHT.[455] As this ***571** review has described, there is a clear, strong, and highly statistically significant association of SDHs and RHs with trauma.[456] However, the mere presence alone of SDHs and RHs does not establish a diagnosis of AHT.

A thorough evaluation, which includes, at a minimum, a complete medical history and physical examination, is required to rule out other causes for the findings. A multidisciplinary approach that involves careful review of psychosocial and investigative details is ideal. Akin to the well-establish medical diagnosis of battered child syndrome, AHT also finds its foundation in "the degree and type of injury [that] is at variance with the history given regarding the occurrence of trauma."[457]

Arriving at the diagnosis is no different than arriving at any other clinical medical diagnosis: it starts with a "chief complaint." In the context of AHT, usually this comprises a presenting symptom or symptoms, such as apnea (stopping breathing), irritability, change in mental status, seizures, lethargy, vomiting or others.[458] With that initial presenting symptom(s), a clinical provider will obtain a comprehensive medical history. This includes a detailed history of the events surrounding the presenting symptom(s), a trauma history, a history of infectious symptoms or exposures, a detailed past medical history (including prior illnesses, surgeries, hospitalizations, and birth history, if applicable), a developmental history, a history of relevant family medical illnesses/disorders, and a comprehensive psychosocial history (including identification of psychosocial stressors, preexisting or concurrent mental health disorders, substance abuse, domestic violence, and prior concerns for child maltreatment/neglect).[459] Typically, this history is obtained by asking the caregiver open-ended, non-suggestive questions, such as: "What happened/did you do next?" or, "How did the infant/child act then/thereafter?" or, "Tell me about your child's ***572** daily activities in the days prior."[460]

Subsequent to the history, the clinical provider conducts, when applicable, a detailed, entire-body physical examination.[461] Special attention is paid to the head, skin, and abdominal, genitourinary, and skeletal systems to assess for signs of trauma.[462] Although the physical examination is an important part of the diagnostic process, historical reports and recent studies have confirmed the absence of any physical findings of trauma on exam in upwards of 31% of AHT cases.[463]

After obtaining a history and performing a physical examination, the clinician considers the various diagnoses that might explain the clinical presentation.[464] This is also known as the "differential" (list of possible causes).[465] The clinician will formulate differentials for all the relevant injuries. For the limited purposes of this article, the most common injuries involved in AHT--SDHs and RHs--have been considered. When presented with the differentials for those injuries (listed in Appendix B and C), the clinician then goes through the complex inferential and deductive process of differential refinement.

Whereas this clinical methodology was once believed to be a linear, Bayesian analysis, it is now understood that the diagnostic process is a dynamic, non-linear, unstructured method of problem-solving.[466] Consequently, and especially in AHT cases, the clinician engages in a multi-disciplinary process of attaining additional ***573** information.[467] The clinician cooperates with multiple agencies (social services and law enforcement) and multiple medical disciplines (radiology, ophthalmology, neurosurgery, etc.) to obtain additional history and clinical information.[468] Furthermore, the clinician examines existing laboratory and radiologic data, and determines the necessity of additional laboratory and/or radiologic testing.[469] Once having

received the additional information, the clinician synthesizes that information with the known pathophysiologic processes of the human body, the evidence-based statistical information on the injuries, and the clinician's own experience in patient care. [470]

For SDHs and RHs, many of the potential disorders on the differential can be eliminated through a detailed history, physical examination, and initial laboratory and radiologic information. [471] In the vast majority of cases, the common denominator for SDHs and RHs will be trauma. [472] From there, the clinician must determine whether the clinical information is consistent with either accidental trauma or AHT. [473]

In arriving at that determination, the clinician closely examines the historical information for consistency. Inconsistency can appear in a variety of ways. The history provided for the injury may have internal features to the story, which are inconsistent with themselves. A history may substantially evolve or change as it is told to multiple providers. Other examples of inconsistency include: 1) a history that is absent in the presence of severe injuries; 2) a history that is inconsistent with the known developmental capabilities of the child; 3) a history that is inconsistent, pathophysiologically, [474] with ***574** the injuries; or 4) a history that is inconsistent with the extensive clinical studies and statistical information (described in the section above, and in Appendix A on SDHs and RHs). As has long been validated, both medically and legally, through the diagnosis of battered child syndrome, if a clinician determines the injuries are "at variance with the history given regarding the occurrence of trauma," [475] then the clinician can diagnose AHT/non-accidental trauma with a reasonable degree of medical certainty.

D. "A Shifted Consensus?"

As mentioned above, recent authors and cases have cited "a shift in mainstream medical opinion" against the validity of AHT as a medical diagnosis. [476] Other proffers have included: "[a]nd as technology and scientific methodology advanced, researchers questioning the basis for SBS reached a critical mass." [477] There is but one simple question for these assertions: Where is the evidence/data for these assertions (other than the opinions of known defense experts)?

Rather than respond in like, with unsupported generalizations, this author will simply cite, with supporting, verifiable references, the various international and domestic medical organizations that have publicly acknowledged the validity of AHT as a medical diagnosis: [478]

1) The World Health Organization [479]

2) The Royal College of Paediatrics and Child Health [480]

***575** 3) The Royal College of Radiologists [481]

4) The Royal College of Ophthalmologists [482]

5) The Canadian Paediatric Society [483]

6) The American Academy of Pediatrics [484]

7) The American Academy of Ophthalmology [485]

8) The American Association for Pediatric Ophthalmology and Strabismus [486]

9) The American College of Radiology [487]

10) The American Academy of Family Physicians [488]

11) The American College of Surgeons [489]

12) The American Association of Neurologic Surgeons [490]

*576 13) The Pediatric Orthopaedic Society of North America [491]

14) The American College of Emergency Physicians [492]

15) The American Academy of Neurology [493]

While it is certainly true that the public promulgations of the various international and domestic medical societies are not representative of each and every member of that society, it is safe to conclude they are representative of the majority of its members. The notable subspecialties that have some discord amongst their members are pathologists (represented by the National Association of Medical Examiners) and biomechanical engineers.

III. The Daubert Analysis and Beyond

A. The Daubert Analysis

A Daubert/Trilogy scrutiny of AHT evidence/testimony can only begin at one place: Daubert. The Daubert court stated that when faced with a proffer of scientific testimony, "the trial judge must determine at the outset, pursuant to Rule 104(a), whether the expert is proposing to testify to (1) scientific knowledge that (2) will assist the trier of fact to understand or determine a fact in issue." [494] These are well-recognized as the reliability and relevance requirements of the trial judge's gate-keeping responsibilities.

In assessing reliability, the Daubert court clearly stated there is *577 no checklist or specific test. However, in assessing the validity of the methodology underlying the proposed scientific testimony, the court enunciated four factors for the trial judge to consider:

1) whether a theory or technique could be (and had been) tested--also known as "falsifiability" or "testability;" [495]

2) "whether the theory or technique had been subjected to peer review and publication;" [496]

3) whether there was a "known or potential rate of error;" [497] and

4) whether there was "general acceptance" in the relevant scientific community. [498]

These four factors will be the starting point of our analysis.

The first two factors, the falsifiability of AHT and its subjection to peer review, are readily addressable. As has been demonstrated above, AHT has been tested or subjected to the scientific rigors of falsifiability by multiple disciplines and multiple methods.[499] Pediatricians, specifically those specializing in child abuse and neglect, have, over many years, studied and tested various facets of AHT diagnosis, such as symptom presentation, historical factors, physical examination findings, laboratory and radiologic findings, and outcomes.[500] Radiologists have utilized imaging modalities (CT and MRI) to assess the frequency and specificity of certain intracranial injuries, like SDHs, in traumatic and non-traumatic scenarios.[501] Biomechanical engineers have examined AHT from ***578** primarily a "physical forces" perspective, seeking to exact quantifiable answers to the forces required to cause the intracranial and spinal injuries seen in AHT.[502] And, finally, pathologists have comparatively studied the microscopic and macroscopic tissue manifestations of the intracranial, intraocular, and spinal injuries in accidental and AHT cases.[503]

But not only has AHT been studied in multiple disciplines and by multiple methods, it also has been studied by multiple researchers from multiple nations. As has been discussed above, there exist at least 700 peer-reviewed, clinical medical articles, comprising thousands of pages of medical literature, published by over 1000 different medical authors, from at least twenty-eight different countries.[504] Additionally, AHT has been peer-reviewed and published in the following disciplines: biomechanical engineering, general pediatrics, neonatology, neurology, neurosurgery, nursing, obstetrics, ophthalmology, orthopedics, pathology (forensic pathology), radiology, and rehabilitative medicine.[505] In fact, given its association with significant medical injuries and child fatalities, AHT is the most peer-reviewed and well-published topic in child abuse pediatrics. Thus, it is difficult for one to assert or argue that the diagnosis of AHT has not been subjected to the rigors of scientific falsifiability, stringently peer reviewed, or well published.

The third criterion--the known or potential rate of error--is Daubert's reference to statistical evidence either in support of or against a particular theory.[506] While certain scientific disciplines have a readily computable error rate, certain scientific disciplines do not. In clinical medical studies, the best approximation of an error ***579** rate is the p-value. Remembering the general statistics section above, the p-value is the probability that the result obtained is secondary to chance.[507] Although chance is not per se error, in clinical medical studies, it is the best approximation, and the cut-off or threshold, for determining what data is reliable.

As discussed above, there are numerous systematic reviews, controlled trials, and well-designed, prospective, and retrospective studies that demonstrate a highly significant statistical association of SDHs and RHs with AHT. For example, recent studies and systemic reviews have calculated the specificity and positive predictive value of severe RHs for abusive head injury to be on the order of 93-97% and 71-96%, respectively.[508] In fact, Vinchon et al. recently determined that the concurrence of these factors--SDH, RH, and the absence of evidence of impact to the head--was 100% specific for abusive injury.[509]

In order to truly appreciate the strength of this statistical evidence, we must, at this point, discuss the concept of "convergent validation."[510] Simply stated, "convergent validation" is the confirmation of a relationship of variables when that relationship is demonstrated by multiple independent measures.[511] The higher ***580** these independent measures correlate with each other, the greater the validity of the results.[512] With SDHs and RHs, the concept of convergent validation explains the increased statistical strength and validity of their results. Both injuries have been studied by multiple independent measures--general pediatrics studies, radiology studies, and pathology studies--and all independent measures have correlating results. Thus, the medical literature on AHT has also addressed Daubert's third criterion.

Finally, with regards to general acceptance within the relevant scientific community criterion, there are several issues that warrant further discussion. First, in the field of AHT, what constitutes the "relevant" scientific community? Is it general pediatricians? Pediatricians who specialize in child abuse and neglect? Pathologists? Ophthalmologists? Second, what constitutes "general acceptance" within that community? Is it a majority of members, or is unanimity or near unanimity required?

Third, how is appropriate evidence of general acceptance adduced? Is the opinion testimony of one random member sufficient? Or is something more definitive required, such as opinion results of a majority of members or a policy statement promulgated by a medical society? Finally, what is the appropriate course of action when multiple disciplines are involved, as in AHT (general pediatrics, radiology, ophthalmology, neurosurgery, and occasionally pathology), and each are relevant scientific communities? Can a specialist from one discipline testify to scientific evidence from the other disciplines?

Although many courts, U.S. and international, have concluded that AHT is a generally accepted valid medical diagnosis [513] within ***581** the relevant scientific community, they have offered little guidance on what the relevant scientific community or general acceptance is and how those determinations came to be. With regards to AHT, the relevant scientific community should be those medical providers who, within their discipline, spend a reasonable portion if not majority, of their clinical time and practice in the evaluation and care of children suspected of AHT and abuse, who remain abreast of the most recent peer-reviewed literature on AHT and child abuse, and who either have obtained subspecialty certification, or are eligible for subspecialty certification, in the field of child abuse. [514] The satisfaction of these criteria will aid a court in assuring that the testimony provided is tethered to standards of medical practice, thereby satisfying Kumho. [515]

The clinical practice of evaluating and caring for children suspected of AHT and abuse is a crucial element in the determination of the relevant scientific community. There are medical subspecialists (general pediatricians, pathologists, radiologists, ophthalmologists, etc.), and even non-medical persons (biomechanical engineers), who are well versed and well read on the literature surrounding AHT. But, a mere reading knowledge of a particular topic cannot be considered relevant to the scientific community. Experiential knowledge is commensurate, if not superior, to didactic knowledge. As the U.K. High Court stated in a recent appeal of shaken baby syndrome cases:

> The fact that an expert is in clinical practice at the time he makes his report is of significance. Clinical practice affords experts the opportunity to maintain and develop their experience. . . . Clinicians learn from each case in which they are engaged. Each case makes them think and as their experience develops so does their understanding. Continuing experience gives them the opportunity to adjust previously held opinions, to alter their views. . . . Such clinical experience . . . may provide a far more reliable source of evidence than that provided by those who have ceased to practice their expertise in a continuing ***582** clinical setting and have retired from such practice. Such experts are, usually, engaged only in reviewing the opinions of others. They have lost the opportunity, day by day, to learn and develop from continuing experience. [516]

Thus, those providers who, in their discipline, do not spend a reasonable portion of their practice in the evaluation and care of AHT and child abuse patients cannot be considered the relevant scientific community within the meaning of Daubert and Kumho.

Courts have historically relied upon opinion testimony to provide evidence of the general acceptance of AHT within the scientific community. [517] Since there is no medical or scientific literature assessing the opinions of physicians on the validity of AHT as a medical diagnosis, a concern with prior opinion testimony on general acceptance is that its foundation may have rested upon the ipse dixit of the expert. Consequently, as expert opinions on the general acceptance of AHT occasionally varied from location to location, and from time to time, so have some court decisions. [518]

Although there is no medical or scientific study assessing the opinions of physicians on the validity of AHT, there is still substantive evidence to that effect--the public promulgations of the relevant national and international medical societies. The very raison d'etre of national and international medical societies is to represent the professional interests of the individual members within those societies. As such, these national and international medical societies have inherent, formal processes for obtaining individual member input on relevant professional topics, considering that input and the relevant scientific literature, and then formulating policy statements, practice guidelines or other educational materials on those topics. ***583** While not

representative of each and every member of that society, it is safe to conclude that the promulgations of the national and international medical societies are at least representative of the professional views of a majority of its members.

With that said, it is virtually unanimous among national and international medical societies that AHT is a valid medical diagnosis.[519] Amongst clinical practitioners, from pediatricians to radiologists, from the American Academy of Pediatrics to the World Health Organization, the validity of AHT as a medical diagnosis is unquestioned. Thus, the fourth Daubert criterion has also been addressed.

Although the four general considerations enunciated in Daubert are satisfied by the AHT literature, the trilogy makes clear that, overall, it is the methodology that is of paramount importance, not the conclusions generated or the criterion satisfied. Does the AHT expert have "good grounds"[520] for coming to his/her conclusions? Is there a logical nexus between his/her methodology and the opinions that are generated? Has the expert exercised the "same level of intellectual rigor"[521] that the expert would use outside the courtroom when working in his/her relevant discipline? Or is AHT just junk science that's not "even good enough to be wrong"[522] and thus inadmissible scientific testimony/evidence?

In assessing the methodology in AHT, it is important to remember that arriving at the diagnosis of AHT employs no different methodology than arriving at any other clinical diagnosis. At its core, clinical medical decision-making is grounded in the roots of the scientific method. Extensive study into physician cognition has revealed valuable insights into the clinical diagnostic process (the methodology sought to be evaluated by Daubert). Whereas it was once thought that physician clinical reasoning proceeded in a ***584** discretely linear fashion known as Bayesian analysis,[523] recent research has demonstrated the diagnostic process is actually a non-linear, unstructured method of problem-solving that employs both inferential and deductive reasoning.[524]

The physician gathers information on a patient's symptoms and signs and generates hypotheses (also known as a differential diagnosis).[525] Through the attainment of additional clinical information (via various diagnostic tests), the physician goes through an inferential and deductive process of hypothesis refinement until a consistent "working diagnosis" is achieved.[526] Hypothesis refinement utilizes a variety of reasoning strategies--probabilistic, causal and deterministic--to discriminate among the existing diagnoses of the differential diagnosis.[527] While being ***585** mindful of the pitfalls of heuristics, the physician ultimately proceeds to hypothesis confirmation when the laws of diagnostic adequacy, coherency, and parsimony are satisfied.[528]

Many courts have held that the "differential diagnosis" methodology is a reliable method of ascertaining medical causation.[529] Courts have stated that the "differential diagnosis is a well-recognized and widely-used technique in the medical community to identify and isolate causes of disease and death."[530] As long as the expert "at least considers alternative causes," then testimony based upon the "differential diagnosis" methodology is admissible.[531]

U.S. courts have previously assessed the methodology underlying AHT and deemed it valid.[532] In more recent cases, U.S. courts have reassessed its sufficiency, and have still deemed it ***586** valid.[533] But, the assessment of the validity of the methodology underlying AHT is not peculiar to U.S. courts.

In the United Kingdom, AHT has been a topic of significant medico-legal concern recently. The U.K. High Court recently heard four appeals on alleged "battered babies" cases.[534] In R v. Harris (a consolidation of the four appeals) the U.K. High Court examined the issue of whether newly-developed "medical research . . . [had created] 'fresh evidence' which . . . [cast] doubt on the safety of each conviction."[535] The High Court stated:

> At the heart of these appeals . . . was a challenge to the accepted hypothesis concerning "shaken baby syndrome" (SBS); or, as we believe it should be more properly called, non-accidental head injury (NAHI). The accepted hypothesis depends on findings of a triad of intracranial injuries consisting of encephalopathy (defined as disease of the brain affecting the brain's function); subdural haemorrhages (SDH); and retinal haemorrhages (RH). [536]

In evaluating the sufficiency of the "triad," the High Court received testimony from over twenty international experts in the field of AHT--"ten medical expert witnesses called on behalf of the appellants and eleven called on behalf the Crown . . . [and] written evidence of four further witnesses." [537] As a part of its examination of the "newly-developed research," the High Court studied Dr. Geddes' Unified Hypothesis:

Between 2000 and 2004 a team of distinguished doctors led by Dr Jennian Geddes, a neuropathologist with a speciality in work with children, produced three papers setting out the results of their research into the triad. In the third paper "Geddes III", the team put forward a new hypothesis, "the unified hypothesis," which challenged the supposed infallibility of the triad. . . .

***587** When Geddes III was published it was, and still is, very controversial. . . . However, early on in the hearing it became apparent that substantial parts of the basis of the unified hypothesis could no longer stand. Dr Geddes, at the beginning of her cross-examination, accepted that the unified hypothesis was never advanced with a view to being proved in court. . . . Further, she accepted that the hypothesis might not be quite correct; or as she put it: "I think we might not have the theory quite right. I think possibly the emphasis on hypoxia--no, I think possibly we are looking more at raised pressure being the critical event." [538]

In concluding that Geddes' Unified Hypothesis could no longer be considered credible, the High Court stated:

As a result of critical papers published in the medical journals, as we have already stated, Dr Geddes when cross-examined frankly admitted that the unified hypothesis could no longer credibly be put forward. In cross-examination she accepted that she could no longer support the hypothesis that brain swelling was the cause of subdural haemorrhages and retinal haemorrhages. She did, however, state that she believed that raised intracranial pressure (ICP) might prove to be an independent cause of both lesions. When asked by Mr Horwell if she had published a paper on this hypothesis she said that she had not and that her research was still incomplete. . . . "In our judgment, it follows that the unified hypothesis can no longer be regarded as a credible or alternative cause of the triad of injuries. . . .

. . . These four appeals raise different medical issues and do not necessarily fail because the unified hypothesis has not been validated. But it does mean that the triad, itself a hypothesis, has not been undermined in the way envisaged by the authors of Geddes III. [539]

The High Court then conducted "sufficiency of evidence" reviews on the four cases. [540] Based upon an appellate standard of review of "whether the evidence, if given at the trial, might reasonably have affected the decision of the trial jury, [541] "the High Court determined that, in two cases, the "fresh" evidence "might reasonably have affected the jury's decision to convict" [542] and set aside those ***588** convictions. [543] In the two other cases, the High Court sustained or modified the convictions. [544]

B. Other Legal Challenges to AHT

Although a comprehensive examination of all the challenges surrounding AHT testimony and evidence is beyond the scope of this article, a couple of more recent challenges shall be addressed briefly. [545] One, akin to Geddes' Unified Hypothesis, is an assertion of an alternative explanation for the injuries seen in AHT. It is the abovementioned "dural immature vascular plexus"

theory by Squier and Mack.[546] This theory is but another example of a more general, overarching challenge to the medical evidence base underlying AHT. By proffering another valid scientific explanation for the injuries in AHT, the contention is that there will then be doubt regarding the "non-accidental," "abusive," and "traumatic" nature of the injuries.

In the dural immature vascular plexus theory, the authors hypothesize that there is a plexus (network) of vessels within the dura mater that is immature and a likely source for "hemorrhage in non-traumatic conditions."[547] Secondary to the immaturity of these vessels, in situations of hypoxia, these vessels "leak," and subsequently result in SDHs.[548] Akin to Geddes' Unified Hypothesis, hypoxic-ischemic injury is the preeminent factor ***589** leading to "hemorrhage in non-traumatic conditions."[549]

This most recent alternative hypothesis for the causation of SDHs and RHs does not survive Trilogy scrutiny. Unlike even Geddes' Unifed Hypothesis, this theory offers no scientific data linking an intradural (within the dura) vascular plexus to the significant subdural hemorrhages seen in AHT.[550] Although published as a review article in a peer-reviewed medical journal, it has not been the subject of any scientific study, in any cohort of patients. Consequently, it has not been tested by the scientific rigors of falsifiability, and has adduced no evidence-based medical literature. Furthermore, by adhering to Geddes' medically and legally discredited theory of hypoxic-ischemic injury as the "unifying" cause for SDHs and RHs, this theory remains outside mainstream medical opinion. Thus, any scientific testimony based upon this theory would be based solely upon the ipse dixit of the expert, and inadmissible under Joiner and Kumho.

Because the theory attempts to perpetuate Geddes' discredited Unified Hypothesis, two recent United Kingdom court opinions have questioned the scientific objectivity of one of its authors, Dr. Squier. In a U.K. family court opinion, the court stated:
Both Dr. Cohen and Dr. Squier subscribe to the Geddes III hypothesis in one form or another. Put at its simplest, each are of the view that hypoxia in children can lead to subdural haemorrhages and retinal haemorrhages in the absence of trauma.

. . . .

. . . They go against the mainstream of current thinking and the analysis of the Court of Appeal in R v. Harris. . . .

. . . .

Dr. Cohen and Dr. Squier support Geddes III, even though Dr. Geddes herself in Harris withdrew from her own unified hypothesis. . . .

In considering the evidence of Dr. Cohen and Dr Squier, I remind myself that four years have passed since Dr. Geddes accepted that her unified hypothesis could no longer credibly be put forward. . . .

***590** I have to consider whether or not these experts have "developed a scientific prejudice" or whether they are in the vanguard of research and learning.[551]

The court then concluded:
I do not doubt the commitment of Dr. Squier and Dr. Cohen to the advancement of the understanding of Shaken Baby Syndrome. As already indicated, I make no criticism and, indeed, it would be wrong to do so, of the fact that neither of them hold mainstream views. There is a significant fundamental difference between academic theories and hypotheses, on the one hand, and the rigorous forensic analysis which is required in care proceedings . . . .

Dr. Squier and Dr. Cohen, I find with regret, have each fallen into that category of expert identified by Butler-Sloss P. in Re LU & LB, namely the expert who has developed a scientific prejudice. As a consequence, I accept the submission of the Local

Authority that Dr. Squier has permitted her convictions to lead her analysis. . . . [E]ach of the significant factual errors made by her served to support her hypothesis of choking and hypoxia.

The overwhelming preponderance of evidence in this case is to the effect that, as of today, medical opinion is that hypoxia does not lead to subdural haemorrhages and retinal haemorrhages . . . .[552]

When Dr. Squier provided testimony in a recent criminal appellate matter, the U.K. High Court stated:
Dr Squier's stance, in oral evidence before us, casts significant doubt upon the reliability of the rest of her evidence and her approach to this case. It demonstrates, to our satisfaction, that she was prepared to maintain an unsubstantiated and insupportable theory in an attempt to bolster this appeal.

. . . .

In the light of our view as to the quality of Dr Squier's evidence before us we conclude it is not capable of undermining the safety of the verdict. For those reasons, we reject the application to call fresh evidence.[553]

***591** The other recent challenge to the admissibility of AHT testimony asserts that a physician's diagnosis of "abusive" or "inflicted" injury is an "improper comment on the mens rea" element of an offense and consequently, an improper "invasion of the province of the jury."[554] In medicine, physicians routinely diagnose intentional acts of patients that result in medical problems. For example, in eating disorders such as bulemia (binge and purge type) and anorexia nervosa, the patient's intentional acts of either purging food recently eaten (bulemia) or not eating food (anorexia) so as to not gain weight are key diagnostic features of those disorders. Many other medical diagnoses--self-cutting behavior, trichotillomania (hair pulling), and illicit substance abuse, to name a few--exist where primary care physicians, in the routine course of clinical medical practice, diagnose intentional acts of patients as key components of medical disease. Additionally, pathologists (specifically forensic pathologists and medical examiners) are routinely called upon to determine intent in the manner and cause of death. And, psychiatrists are sometimes requested to determine an individual's capacity to satisfy the mens rea elements of criminal offenses. The practice of child abuse pediatrics is no different than these other practices of medicine.

Courts have long held that, as long as a physician does not testify to the ultimate question of the defendant's guilt or innocence, a physician may opine that injuries are "nonaccidental," "inflicted," or "abusive."[555] In Estelle v. Maguire the U.S. Supreme Court recognized the admissibility of medical testimony on the issue of intent when it considered the admissibility of 404(b) evidence in ***592** order to prove "battered child syndrome."[556] The Supreme Court wrote:
The demonstration of battered child syndrome "simply indicates that a child found with [serious, repeated injuries] has not suffered those injuries by accidental means." Thus, evidence demonstrating battered child syndrome helps to prove that the child died at the hands of another and not by falling off a couch for example, it also tends to establish that the "other," whoever it may be, inflicted the injuries intentionally.[557]

As with battered child syndrome, the non-accidental or abusive determination in AHT finds its diagnostic underpinning in "the degree and type of injury [that] is at variance with the history given regarding the occurrence of the trauma."[558] Recently, in State v. Torres, the Supreme Court of Kansas concluded that a physician's opinion that an infant's death was a "textbook case" of "shaken baby or shaken impact syndrome" did not invade the province of a jury so long as the expert did not testify as to "the ultimate question of the defendant's guilt or innocence."[559] Thus, these most recent challenges to the admissibility of AHT testimony lack legal and medical foundation.

C. Beyond Daubert: The Marriage of Medical and Legal Perspectives

Given the abundance of medical literature in support of AHT--the significant statistical strength of much of that literature, the recognition by many U.S. and U.K. courts of the validity of that literature and of the diagnosis of AHT--one must seek explanation for the variability in some court decisions. Why have some courts concluded that there is a "significant and legitimate debate in the medical community" on AHT,[560] while others have not?[561] Why ***593** have some concluded that the diagnosis of AHT is "based on inconclusive research,"[562] while the vast majority have not?[563] Several reasons exist.

First, as mentioned above, the adduction of evidence on what is general acceptance within the relevant scientific community has in many cases, unfortunately, been upon the ipse dixit of the expert. In State v. Edmunds, the Court determined, based upon "expert medical testimony," that "a significant and legitimate debate in the medical community has developed in the past ten years" on AHT.[564] However, those "experts" provided no substantive medical literature affirming that "significant and legitimate debate."[565] Highlighting the shortcomings of such evidence, one expert witness in a U.K. AHT case stated:
Al-Sarraj told the court that there are 40-44 neuropathologists in the country of whom a maximum of 10 or 12 are forensic neuropathologists. To his knowledge, the only neuropathologist in the UK believing that hypoxia can cause subdural haemorrhages is Dr. Waney Squier. In addition, he said there are two or three other people who share her opinion who are working in different, but related, specialities, of whom Dr. Cohen and Dr. Scheimberg (Dr. Cohen's co-author) are presumably two. Dr. Al-Sarraj said:
"They come in all the defence cases, so you do not realise that they are in such a minority."[566]

Second, the pecuniary interest in providing expert testimony cannot be underestimated. It has posed and continues to pose a significant risk to the presentation of unbiased medical information. Third, in addition to pecuniary interest, as discussed above, personal prejudices can also affect scientific analysis. This can result ***594** in the adherence to disproven theories and the presentation of skewed information. Finally, the increasing complexity of scientific and medical information has placed onerous burdens on the single, gate-keeping trial judge. Given the lack of dispositive medical guidance from a unified, unbiased, multi-disciplinary, medical body, courts have been left to fend for themselves, relying upon whatever seemingly reliable medical information is presented. Naturally, variability in some decisions has ensued.

If the marriage of the legal and medical perspectives is to survive, especially with regards to AHT, then the medical and legal fields must remain faithful to their obligations, and seek to strengthen their union. Courts must remember Justice Breyer's admonition--"seek decisions that fall within the boundaries of scientifically sound knowledge"[567] and keep out science that "isn't even good enough to be wrong."[568] This article has provided evidence-based medical literature supporting the scientific soundness of AHT and the lack of such evidence for theories such as Geddes' Unified Hypothesis and Squier and Mack's dural immature vascular plexus theory. Concurrent with that obligation, courts must recognize when there is a legitimate and responsible disagreement among medical experts, and allow the jury to resolve that dispute among the experts. Finally, when confronted with the complexities of medical and scientific information, courts should seek assistance from impartial court-appointed scientific experts to explain the medical and scientific information.

For medicine's part, the national medical societies of the relevant disciplines should coordinate with Federal Judicial Center (FJC) and National Academy of Sciences, Committee on Science, Technology and Law, to establish a registry of potential independent medical experts on AHT. Along those lines, the relevant national medical societies should promulgate policies limiting expert medical testimony fees, and support state and federal legislation towards that effect. Finally, the judiciary, via the FJC, and the relevant medical disciplines, specifically child abuse ***595** pediatricians, should engage in reciprocal educational efforts on the responsibilities and limitations of expert testimony in AHT.

IV. Conclusion

What has been presented for the reader is:
i) a brief examination of the extensive clinical medical literature on the topic of AHT;

ii) evidence-based clinical medical studies on SDHs and RHs that demonstrate highly significant statistical associations of those injuries with AHT;

iii) verifiable references to fifteen national and international medical societies who have publicly endorsed the validity of AHT;

iv) medical and legal rationales refuting alternative hypotheses (such as Geddes' Unified Hypothesis and Squier and Mack's Dural Immature Vascular Plexus Theory) for the injuries common to AHT; and

v) national and international case law examining, and ultimately confirming, the validity of the medical evidence in support of AHT.

These reasons, and years of clinical experience, are the foundation for the opinions given by the vast majority of medical professionals called to evaluate suspected AHT. The diagnosis of AHT, long recognized as a valid diagnosis, occurs within the same professional culture of science and practice (methodology) that leads to the diagnosis and treatment of millions of pediatric patients in the U.S. every year. Many of these diagnoses are matters of life and death, and sometimes these diagnoses lead to the courtroom. For the legal profession to treat this aspect of pediatric medicine as separate from the rest of medicine is unjustifiable. It is understandable that lawyers will look for opportunities to create doubt in the minds of jurors. However, the only way to appropriately improve the chances for justice in the courts with respect to AHT is to assure that an unbiased, financially-unmotivated, medical expert testifies to the current state of medical evidence.

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Accidents/Falls:

Meta-Analysis/Systematic Reviews/Guidelines:

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2) S.A. Schutzman et al., Evaluation and Management of Children Younger Than Two Years Old with Apparently Minor Head Trauma: Proposed Guidelines, 107 Pediatrics 983 (2001).

Controlled Studies/Trials:

1) M. Belechri et al., Bunk Versus Conventional Beds: A Comparative Assessment of Fall Injury Risk, 56 J. Epidemiology & Community Health 413 (2002).

Comparative Cohorts /Prospective Case Series:

1) Christine T. Chiaviello et al., Stairway-Related Injuries in Children, 94 Pediatrics 679 (1994).

2) Christine T. Chiaviello et al., Infant Walker-Related Injuries: A Prospective Study of Severity and Incidence, 93 Pediatrics 974 (1994).

3) Ann-Christine Duhaime et al., Head Injury in Very Young Children: Mechanisms, Injury Types and Ophthalmologic Findings in 100 Hospitalized Patients Younger than 2 Years of Age, 90 Pediatrics 179 (1992).

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Retrospective Case Series/Case Reports:

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14) L. Morrison et al., Infant-furniture Related Injuries among Preschool Children in New Zealand, 1987-1996, 38 J. Paediatrics & Child Health 587 (2002).

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2) Gina E. Bertocci et al., Influence of Fall Height and Impact Surface on Biomechanics of Feet-First Free Falls in Children, 35 Injury 417 (2004).

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***628 Appendix B**

Differential Diagnosis of Subdural Hemorrhages:

Trauma
Inflicted/Abusive

Accidental

Birth

Metabolic Diseases
Glutaric Aciduria Type I

Menke's Disease

Hemophagocytic Lymphohistiocytosis

Nutritional deficiencies

Genetic Syndromes
Osteogenesis Imperfecta

Ehlers-Danlos Syndrome Type II

Hereditary Hemorrhagic Telangectasia

Coagulopathies (Clotting Disorders)
Hemophilia

Hemorrhagic Disease of the Newborn

Tumors
Lymphoblastic Leukemia

Neuroblastoma

Infections
HSV meningoencephalitis

Bacterial meningitis

***629 Appendix C**

Differential Diagnosis of Retinal Hemorrhages:

Trauma
Inflicted/Abusive

Accidental

Birth

Metabolic Diseases
Glutaric Aciduria Type 1

Hemophagocytic Lymphohistiocytosis

Nutritional deficiencies

Genetic Syndromes
Osteogenesis Imperfecta

Ehlers-Danlos Syndrome Type II

Anemia

Coagulopathies (Clotting Disorders)
Hemophilia

Hemorrhagic Disease of the Newborn

Carbon Monoxide Poisoning

Vasculitis

Hypoxia/Hypo or Hypertension

Papilledema/Increased Intracranial Pressure

Tumors
Lymphoblastic Leukemia

Cerebral Aneurysm

Hemangioma

Infections
HSV meningoencephalitis

Bacterial meningitis

***630 FIGURES**

TABULAR OR GRAPHIC MATERIAL SET FORTH AT THIS POINT IS NOT DISPLAYABLE
Fig. 1. Image of Auguste Ambroise Tardieu (1818-1879).

PD-1923. Image originally from Goupil et Cie, http:// www.biusante.parisdescartes.fr/histmed/image?

CIPC0155, available at http:// en.wikipedia.org/wiki/File:AugusteAmbroseTardieu.jpg.

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Fig. 2. First page of Ambroise Tardieu's Etude medico- legale sur les sevices et mauvais traitements exerces sur des enfants (Forensic study on cruelty and ill treatment of children), 1860. Reprinted from Ambroise Tardieu, Etude Medico-Legale sur les Sevices et MauvaisTraitements Exerces sur des Enfants, 13 Annales D'hygiène Publique et de Médecine Légale 361-98 (1860))

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Fig. 3. Image of Wilfred Batten Lewis Trotter (1872-1939). Reproduced with permission © Godfrey Argent Studio.

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***631** Fig. 4. Dr. C. Henry Kempe. Reprinted with permission of The Kempe Foundation for the Prevention and Treatment of Child Abuse and Neglect.

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Fig 5. First page of The Battered-Child Syndrome. JAMA Vol.181 July 7, 1962, pp.17-24. Copyright © 1962 American Medical Association. All rights reserved. Reprinted with permission from JAMA.

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Fig. 6. Human Eye Reprinted courtesy of http://lhsanatomy4.wikispaces.com

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Fig. 7. Normal Retina, demonstrating the area of the retina called the posterior pole: fovea and macula (within circles), optic nerve (bright whitish appearing circle on left-hand side) and its head manifesting as a circular disc (optic disc), and retinal vessels emanating from the optic nerve. Reprinted from Eye Disease Anatomy, Ref#: EDA06, Nat'l Eye Inst., http:// www.nei.nih.gov/ phot/eyedis/index.asp (circles added by author).

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*632 Fig. 8. Mild nonspecific retinal hemorrhages confined to the posterior pole.

(Courtesy of Alex V. Levin, MD. MHSc, Wills Eye Institute, Philadelphia)

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Fig. 9. Severe retinal hemorrhages, too numerous to count, such that there is virtually no visible normal retina. (Courtesy of Alex V. Levin, MD. MHSc, Wills Eye Institute, Philadelphia)

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*633 Fig. 10. Macular traumatic retinoschisis. (Courtesy of Alex V. Levin, MD. MHSc, Wills Eye Institute, Philadelphia)

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Fig. 11. "Retinal hemorrhage in the AT and IHI groups. Although most cases of abuse were associated with severe hemorrhage, seven had no hemorrhage, and three had only mild hemorrhages." Matthieu Vinchon et al., Confessed Abuse Versus Witnessed Accidents in Infants: Comparison of Clinical, Radiological, and Ophthalmological Data in Corroborated Cases, 26 Child's Nervous Sys. 637, 641 fig.3 (2009). Conversely, no or mild RHs were found in 34 cases of AT, id. at 639, 641 fig.3, and only "one had severe hemorrhage caused by direct facial impact." Id. at 641 fig.3. (Figure reprinted with permission of publisher.)

*635 Please stay tuned for an academic response to A Daubert Analysis of Abusive Head Trauma/Shaken Baby Syndrome in volume 12 of this journal.

Footnotes

a1 I am indebted to many for their invaluable assistance in the creation of this document. However, some bear specific recognition. I would specifically like to thank Dr. Betty Spivack, Dr. Chris Greeley, Dr. Alex Levin, Dr. Andy Sirotnak, Dr. Antonia Chiesa, and, most importantly, my friend and mentor, Dr. Don Bross. This article is not only a brief synopsis and testament to the diagnostic genius of our clinical forefathers (Tardieu, Trotter, Caffey, Silverman, Kempe, Guthkelch, and others), but is a salutation of respect and admiration for ALL multidisciplinary colleagues throughout the country who continue to strive for safe, just and equitable outcomes for abused children and their families.

1 This term was one of the earliest descriptive terms of Abusive Head Trauma coined by Dr. John Caffey (often referred to as the Father of Pediatric Radiology). John Caffey, On the Theory and Practice of Shaking Infants. Its Potential Residual Effects of Permanent Brain Damage and Mental Retardation, 124 Am. J. Diseases Child. 161, 161-69 (1972).

2 This is not to minimize the recent important shift towards more accurate terminology in describing this medical diagnosis. As noted by one prominent author, "semantic choices play a large role in how concepts spread, are challenged, and evolve. Sometimes what

we call something hinders our ability to observe all the available facts clearly and come to a more correct or more encompassing understanding of a particular disease process." See Ann-Christine Duhaime, Calling Things What They Are, 3 J. Neurosurgery: Pediatrics 472, 472 (2009).

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4 See Deborah Tuerkheimer, The Next Innocence Project: Shaken Baby Syndrome and the Criminal Courts, 87 Wash. U. L. Rev. 1, 1 (2009); see also Molly Gena, Shaken Baby Syndrome: Medical Uncertainty Casts Doubt on Convictions. 2007 Wis. L. Rev. 701, 718 (2007).

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6 See Cavazos v. Smith, 132 S. Ct. 2, 10 (2011) (per curiam) (Ginsburg, J., dissenting); State v. Edmunds, 746 N.W.2d 590, 596 (Wis. Ct. App. 2008) (granting the defendant/appellant a new trial on the basis defendant presented "newly discovered evidence" of a "significant and legitimate debate in the medical community" regarding Shaken Baby Syndrome, which has emerged in the past ten years); Order Determining Admissibility of Expert Testimony on AHT/SBS at 22-23, Commonwealth v. Davis, No. 04-CR-205 (Ky. Cir. Ct., Apr. 17, 2006); Tuerkheimer, supra note 4, at 36 (citing State v. Hyatt, No. 06M7-CR00016-02, (Mo. Cir. Ct Nov. 6, 2007) ("[T]he SBS diagnosis 'appears to have gained considerable acceptance... among pediatricians. However, there is substantial, persistent and continuing criticism of this diagnosis among many in the medical and scientific research communities.'"). The American Academy of Pediatrics Section on Child Abuse and Neglect has recently issued a policy statement recommending the use of a more accurate, and less mechanistically constricting, term of "Abusive Head Trauma." See Cindy W. Christian et al., Abusive Head Trauma in Infants and Children, 123 Pediatrics 1409, 1410-11 (2009). Consequently, for the remainder of this article I will refer to the concept of Shaken Baby Syndrome as "Abusive Head Trauma."

7 See Tuerkheimer, supra note 4, at 22.

8 Id. at 11.

9 Id. at 12.

10 Id. at 12-13; see also Gena, supra note 4, at 720.

11 Tuerkheimer, supra note 4, at 14; see also Gena, supra note 4, at 710.

12 The American Academy of Pediatrics provides a general assessment of the topic, but its purpose was not intended to be a critical analysis of the literature on the topic. American Academy of Pediatrics, Shaken Baby Syndrome: Rotational Cranial Injuries--Technical Report, 108 Pediatrics 206, 206 (2001). One other article has been proffered, and frequently cited by opponents of Abusive Head Trauma, to be a critical review of the literature on the topic. Mark Donohoe, Evidence-Based Medicine and Shaken Baby Syndrome, 24 Am. J. Forensic Med. & Pathology 239, 239 (2003). A critical evaluation of that article will be conducted in detail herein below.

13 Abusive Head Injury/Shaken Baby Syndrome entails a wide constellation of symptoms and injuries with varying degrees of severity. The most common injuries associated with this diagnosis are intracranial hemorrhage (most commonly subdural or subarachnoid hemorrhage) and retinal hemorrhages. See Antonia Chiesa & Ann-Christine Duhaime, Abusive Head Trauma, 56 Pediatric Clinics N. Am. 317 (2009). While many other injuries are associated with this diagnosis, this paper will focus on the clinical medical literature behind the most common injuries--subdural hemorrhage and retinal hemorrhages. A thorough examination of the literature behind all the possible injuries and all potential causes (short falls, biomechanics of head injury, etc.) is simply too broad and beyond the scope of this paper. For a more comprehensive examination of the literature on this topic, I would reference the reader to Lori Frasier et al., Abusive Head Trauma in Infants & Children: A Medical, Legal, and Forensic Reference (2006). See also Lucy Rorke-Adams et al., Head Trauma, in Child Abuse: Medical Diagnosis & Management 53 (Robert M. Reece & Cindy W. Christian eds. 2009).

14 Frye v. United States, 293 F. 1013, 1014 (D.C. Cir. 1923).

15 Daubert v. Merrell Dow Pharm., Inc., 509 U.S. 579, 589 (1993)

16 Id.

17 Id. at 584-85.

18 Id. at 582.

19 Id.

20 Id. at 583.

21 Id. at 584-85.

22 Id. at 585.

23 Id. at 589; id. at 598 (Rehnquist, C.J., concurring in part and dissenting in part).

24 Id.

25 E.g. Beech Aircraft Corp. v. Rainey, 488 U.S. 153, 169 (1988).

26 Daubert, 509 U.S. at 588.

27 Id. at 594.

28 Id. at 589.

29 Id. at 589-90.

30 Id. at 590 n.9.

31 Id.

32 Id. at 593-94.

33 Id. at 593.

34 Id. at 594-95 (emphasis added).

35 Id. at 591.

36 Id. at 591-92 (emphasis added)

37 Gen. Elec. Co. v. Joiner, 522 U.S. 136, 138-39 (1997).

38 Id. at 141.

39 See Joe S. Cecil, Ten Years of Judicial Gatekeeping Under Daubert, 95 Am. J. Pub. Health s74, s75 (Supp. 2005).

40 Joiner, 522 U.S. at 139.

41 See Cecil, supra note 39, at s76.

42 Joiner, 522 U.S. at 146-47.

43 Id. at 146 (emphasis added).

44 Kumho Tire Co. v. Carmichael, 526 U.S. 137, 141 (1999).

45 Id. at 147.

46 Id. at 149.

47 Id. at 151.

48 See id.

49 Id.

50 See id. at 152.

51 Id. at 150 (quoting Brief for United States as Amicus Curiae Supporting Petitioners at 19, Kumho Tire Co. v. Carmichael, 526 U.S. 137 (1999)). Some legal scholars commented that the Court's decision in Kumho sought to rectify a bias in Daubert towards the "hard sciences" which employ rigorous empirical methods. See Paul S. Milich, Controversial Science in the Courtroom 43 Emory L.J. 913, 917 (1994) ("Daubert... never mentions the psychological sciences, for example, where much of the data is subjective and many of the theories are empirically difficult, if not impossible, to verify"); see also Ralph Underwager & Hollida Wakefield, A Paradigm Shift for Expert Witnesses, Issues in Child Abuse Accusations, Summer 1993, http://ipt-forensics.com/journal/volume5/j5_3_2.htm ("American psychiatry is, by and large, Freudian in its orientation" and "wherever Freudian theory has been subjected to empirical tests, it has either failed, or, at best, been inconclusive as a predictor of human behavior."). Yet psychiatry is a recognized science readily integrated into and accepted by the criminal justice system when issues of mental competency arise.

52 Kumho, 526 U.S. at 152.

53 See Margret Berger, The Supreme Court's Trilogy on the Admissibility of Expert Testimony, in Fed. Judicial Ctr., Reference Manual on Scientific Evidence 9, 21 (2d ed. 2000), www.fjc.gov/public/pdf.nsf/lookup/sciman00.pdf/$file/sciman00.pdf.

54 See Jerome Kassirer & Joe Cecil, Inconsistency in Evidentiary Standards for Medical Testimony: Disorder in the Courts, 288 JAMA 1382, 1383 (2002).

55 Fed. Judicial Ctr., Reference Manual on Scientific Evidence, at v (2d ed. 2000) (quoting T.H. Huxley, The Crayfish: An Introduction to the Study of Zoology 2 (1880)).

56 D. Allen Bromley, Science and the Law, Address at the 1998 Annual Meeting of the American Bar Association (Aug. 2, 1998).

57 Stephen Breyer, Introduction, Fed. Judicial Ctr., Reference Manual on Scientific Evidence 2, 4 (2d ed. 2000), www.fjc.gov/public/pdf.nsf/lookup/sciman00.pdf/$file/sciman00.pdf.

58 Id. at 3.

59 See id.

60 See id.

61 Id.

62 Dep't of Commerce v. U.S. House of Representatives, 525 U.S. 316, 320 (1999); Breyer, supra note 57, at 2.

63 Kansas v. Hendricks, 521 U.S. 346, 350 (1997); Breyer, supra note 57, at 3.

64 Washington v. Glucksberg, 521 U.S. 702, 722 (1997); Vacco v. Quill, 521 U.S. 793, 797 (1997); Breyer, supra note 57, at 3.

65 See The Law Comm'n, Consultation Paper 190, The Admissibility Of Expert Evidence in Criminal Proceedings in England and Wales: A New Approach to the Determination of Evidentiary Reliability, P 2.8 n.6, P 2.28 (2009), www.lawcom.gov.uk/docs/cp190.pdf (discussing how M. Redmayne, in Expert Evidence and Criminal Justice, "summarizes research which suggests that as expert evidence becomes more complicated, jurors shift their focus and rely on peripheral indicia of reliability"); see also id. at P 2.3 (citing Paul Roberts & A.A.S. Zuckerman, Criminal Evidence 292-96 (2004)).

66 See U.S. v. Addison, 498 F.2d 741, 744 (1974) (The Court stated, "scientific proof may in some instances assume a posture of mystic infallibility in the eyes of a jury of laymen"); see also John William Strong, Language and Logic in Expert Testimony, 71 Or. L. Rev. 349, 367-68 n.81 (1992) ("There is virtual unanimity among courts and commentators that evidence perceived by jurors to be 'scientific' in nature will have particularly persuasive effect.").

67 See Stephen T. Goudge, Inquiry into Pediatric Forensic Pathology in Ontario 531 (Ontario Ministry of the Att'y Gen. 2008); see also The Law Commission, Consultation Paper 190, supra note 65, at PP 2.14-2.22 (2009). (citing three recent AHT/SBS cases in England and Wales where criminal convictions were obtained and subsequently overturned on appeal because of "flawed" scientific evidence/testimony). But see Neil Vidmar & Shari Seidman Diamond, Juries and Expert Evidence, 66 Brooklyn L. Rev. 1121, 1179 (2001) ("Empirical data do not support a view that juries are passive, too-credulous, incompetent, and overawed by the mystique of the expert.").

68 Daubert v. Merrell Dow Pharm., Inc., 509 U.S. 579, 590 (1993) (second emphasis added).

69 Bert Black, A Unified Theory of Scientific Evidence, 56 Fordham L. Rev. 595, 599 (1988); see also Bert Black et al., Science and the Law in the Wake of Daubert: A New Search for Scientific Knowledge, 72 Tex. L. Rev. 715, 753 (1994).

70 Clifton T. Hutchinson & Danny S. Ashby, Redefining the Bases of Admissibility of Expert Scientific Testimony, 15 Cardozo L. Rev. 1875, 1886 (1994).

71 Breyer, supra note 57, at 4 (emphasis added).

72 See The Law Comm'n, supra note 65, at P 1.5.

73 Id. at 47.

74 Id. at 49-51.

75 Gen. Elec. Co. v. Joiner, 522 U.S. 136, 146 (1997).

76 See Daubert v. Merrell Dow Pharm., Inc., 509 U.S. 579, 600 (Rehnquist, C.J., concurring in part and dissenting in part). In response to this concern, the Federal Judicial Center, the research and educational arm of the federal judicial system, has published a 1034-page reference source (currently in its third edition) to help federal judges "manage cases involving complex scientific and technical evidence." See Federal Judicial Center, Reference Manual on Scientific Evidence, at xv (3d ed. 2011), http:// www.fjc.gov/public/pdf.nsf/lookup/SciMan3D01.pdf/$file/SciMan3D01.pdf. For other comprehensive references on the issues surrounding Science, Law, and Expert testimony, see generally 1 Modern Scientific Evidence: The Law and Science of Expert Testimony (David L. Faigman et al. eds., 1997); Expert Evidence: A Practitioner's Guide to Law, Science, and the FJC Manual (Bert Black & Patrick W. Lee eds., 1997).

77 Daubert v. Merrell Dow Pharm., Inc., 43 F.3d 1311, 1316 (9th Cir. 1995). However, trial judges have adapted to the heady responsibility of the trilogy decisions by utilizing innovative case-management techniques, such as court-appointed independent experts or court-appointed scientific panels, to assist with the comprehension of complex scientific information. Furthermore, public and private organizations, such as the American Association for the Advancement of Science (AAAS), have offered trial judges the service of locating impartial, skilled experts at fee-for-service costs. See Court Appointed Scientific Experts, Am. Ass'n for the Advancement of Sci., http:// www.aaas.org/spp/case/case.htm (last visited Oct. 21, 2011).

78 Sophia I. Gatowski et al., Asking the Gatekeepers: A National Survey of Judges on Judging Expert Evidence in a Post-Daubert World, 25 L. & Hum. Behav. 433, 442-47 (2001).

79 Id. at 444-45.

80 Id. at 443.

81 Id. at 452.

82 Kassirer & Cecil, supra note 54, at 1384. "This approach was urged by the United States Court of Appeals for the Ninth Circuit when it reconsidered the Daubert case." Id.

83 Id. (citing Jones v. United States, 933 F. Supp 894, 897 (N.D. Cal. 1996)).

84 Id. (citing Raynor v. Merrell Pharm. Inc., 104 F.3d 1371, 1375 (D.C. Cir 1997)).

85 See Haggerty v. Upjohn Co, 950 F. Supp. 1160, 1165 (S.D. Fla. 1996); Hall v. Baxter Healthcare Corp, 947 F. Supp. 1387, 1411 (D. Or. 1996).

86 See Pick v. Am. Med. Sys., Inc., 958 F. Supp. 1151, 1160-62 (E.D. La. 1997); Glaser v. Thompson Med. Co., 32 F.3d 969, 975 (6th Cir. 1994); Cella v. United States, 998 F.2d 418, 426 (7th Cir. 1993).

87 See Cecil, supra note 39, at s76 (citing Newman v. Motorola Inc., 218 F. Supp. 2d 769, 780-81 (D. Md. 2002); Amorgianos v. Nat'l R.R. Passenger Corp., 137 F. Supp.2d 147, 189 (E.D.N.Y. 2001); Mitchell v. Gencorp Inc., 165 F.3d 778, 782 (10th Cir. 1999)).

88 See Cecil, supra note 39, at s75. This data is in contrast to one author's assertion of judicial deference to admissibility of testimony on Abusive Head Trauma/Shaken Baby Syndrome. See Tuerkheimer, supra note 4, at 42-44.

89 See Cecil, supra note 39, at s75.

90 See Kassirer & Cecil, supra note 54, at 1382.

91 See Mary Sue Henifin et al., Reference Guide on Medical Testimony, in Fed. Judicial Ctr., Reference Manual on Scientific Evidence 439, 465 (2d ed., 2000), http://www.fjc.gov/public/pdf.nsf/lookup/sciman00.pdf/ $file/sciman00.pdf; see also Jerome Groopman, How Doctors Think 7 (2007).

92 Mark B. McClellan et al., Evidence-Based Medicine and the Changing Nature of Health Care: 2007 IOM Annual Meeting Summary 94 (Nat'l Acad. of Scis. 2008).

93 Some prefer to refer to this as an "applied science" rather than an "art." See Harriet Hall, The "Art" of Clinical Decision-Making, Science-Based Medicine (May 13, 2008), http://www.sciencebasedmedicine.org/index.php/the-art-of-clinical-decision-making/.

94 See McClellan et al., supra note 92, at 94.

95 See Henifin et al., supra note 91, at 465.

96 See Groopman, supra note 91, at 37; see also Pat Croskerry, The Importance of Cognitive Errors in Diagnosis and Strategies to Minimize Them, 78 Acad. Med. 775, 775 (2003); Pat Croskerry, Achieving Quality in Clinical Decision Making: Cognitive Strategies and Detection of Bias, 9 Acad. Med. 1184, 1184 (2002).

97 Groopman, supra note 91, at 35-36, 39.

98 See Croskerry, The Importance, supra note 96, at 776; Croskerry, Achieving Quality, supra note 96, at 1184.

99 See About Us, The Cochrane Collaboration, http:// www.cochrane.org/about-us (last visited Jan. 24, 2012); Happy 35th Birthday, MedLine!, U.S. Nat'l Library Med., http://www.nlm.nih.gov/news/medline_35th_ birthday.html (last updated Oct. 23, 2006) (showing the Medline database was founded in 1971).

100 See History of Systematic Reviews, EPPI-Centre, http:// eppi.ioe.ac.uk/cms/Default.aspx?tabid=68 (last visited, Jan. 24, 2012).

101 See id.

102 David Sackett, et al., Evidence Based Medicine: What It Is and What It Isn't: It's About Integrating Individual Clinical Expertise and the Best External Evidence, 312 Brit. Med. J. 71, 71 (1996). The determination of what the "current best evidence" is in a given field requires a critical evaluation of the relevant medical literature, utilizing statistical principles to assess the validity of studies and the conclusions they reach. See id. at 72. We will discuss basic principles of statistical analysis herein below when we critically evaluate the "current best evidence" in the field of Abusive Head Trauma. See also McClellan et al., supra note 92, at v.

103 McClellan et al., supra note 92, at v (emphasis added).

104 Id.

105 William W. Stead & John M. Starmer, Beyond Expert-Based Practice, in McClellan et al., supra note 92, at 94.

106 Id. at 94.

107 Controversy exists between the American Cancer Society and the United States Preventive Services Task Force on breast cancer and prostate cancer screening guidelines. For further review, the reader should examine the respective societies' websites.

108 See Kassirer & Cecil, supra note 54, at 1383.

109 Id.

110 Groopman, supra note 91, at 6.

111 George Santayana, The Life of Reason 284 (Charles Scribner's Sons 1905).

112 See Al-Holou et al., supra note 3, at 474.

113 Id. at 475 (citing Ambroise Tardieu, Etude Medico-Legale sur les Sevices et Mauvais Traitements Exerces sur des Enfants, 13 Annales D'hygiène Publique et de Médecine Légale 361-98 (1860)).

114 Id.

115 Id. at 476.

116 Id. at 475.

117 Id.

118 Id.

119 Id. at 476.

120 Id.

121 Id.

122 Id.

123 Id.

124 Id. It is one learned scholar's opinion that use of this terminology constricted the open and comprehensive scientific evaluation of the cause of such injuries in many of the earlier cases, resulting in probable misdiagnosis in many cases. See Duhaime, supra note 2, at 472.

125 Al-Holou et al., supra note 3, at 476.

126 Id.

127 Id. at 477.

128 Id. at 476; see also id. at 481 nn.7, 13, 482 nn.21, 34, 47, 61, 67, 483 nn.74, 86 (citing reports documenting the association of SDHs, ophthalmic hemorrhages, and sometimes bone lesions in infants).

129 See Al-Holou et al., supra note 3, at 478.

130 Id. at 477-78.

131 Id. at 478.

132 Id. (citing Wilfred Trotter, Chronic Subdural Haemorrhage of Traumatic Origin, and Its Relation to Pachymeningitis Haemorrhagica Interna, 2 Brit. J. Surgery, 271-91 (1914)).

133 Id. at 478.

134 Id. at 478 (citing Max. M. Peet & Edgar A. Kahn, Subdural Hematoma in Infants, 98 JAMA, 1851-56 (1932)).

135 Id. at 478-79 (citing Franc D. Ingraham & Donald D. Matson, Subdural Hematoma in Infancy, 24 J. Pediatrics 1-37 (1944)).

136 Id. at 478 (citing Franc D. Ingraham & Henry L. Heyl, Subdural Hematoma in Infancy and Childhood, 112 JAMA. 198-204 (1939)).

137 See id.

138 As will be discussed herein below, this is not to say that these causes (infectious, nutritional, metabolic, etc.) are no longer considered potential causes of SDHs, just that they are no longer considered the primary cause of SDHs. See id.

139 Id. at 479.

140 Al-Holou et al., supra note 3, at 479 (citing John Caffey, Multiple Fractures in the Long Bones of Infants Suffering from Chronic Subdural Hematoma, 56 Am. J. Roentgenology 163-73 (1946)).

141 Paul K. Kleinman & Paul D. Barnes, Head Trauma, in Diagnostic Imaging of Child Abuse, 285, 297 (2d ed. 1998).

142 Al-Holou et al., supra note 3, at 479.

143 Id.

144 Kleinman & Barnes, supra note 141, at 297-98.

145 See Al-Holou et al., supra note 3, at 479 (citing F. Silverman, The Roentgen Manifestations of Unrecognized Skeletal Trauma in Infants, 69 Am. J. Roentgenology Radium Therapy Nuclear Med. 413-27 (1953)).

146 Id.

147 Kleinman & Barnes, supra note 141, at 298 (citing F. Burke, et al., Traumatic Periostitis and Subdural Hematoma, 12 Clinical Procs. Child. Hosp., D.C. 240-46 (1956); P. Josserand et al., Un Nouveau Cas D'Hematome Sous-Dural Associe a des Fractures de Membres Chez un Nourrisson, 15 Pediatrie 647-59 (1960); G. Kinley, et al., Subdural Hematoma, Hygroma, and Hydroma in Infants, 38 J. Pediatrics 667-86 (1951); M.R. Klein, L'Hematome Sous-Dural Du Nourrisson, 21 Archives Francaises de Pediatrie 425-40 (1964); G. Lazorthes, et al., Les Epanchements Sous-Duraux Du Nourrisson: Discussion Etiopathogenique a Propos de 59 Cas, 71 Presse Med. 1903-05 (1963); M. Lelong et al., L'Hematome Sous-Dural Chronique du Nourrisson, 12 Archives Francaises de Pediatrie 1037-84 (1955); E.F. Lis & G.S. Frauenberger, Multiple Fractures Associated With Subdural Hematoma in Infancy, 6 Pediatrics 890-92 (1950); J. Meneghello & J. Hasbun, Hematoma Subdural y Fractura de los Huesos Largos, 22 Revista Chilena de Pediatria 80-83 (1951); N. Neimann et al., Les Enfants Victimes de Services, 23 Pediatrie 861-75 (1968); M.J. Smith, Subdural Hematoma with Multiple Fractures: Case Report, 63 Am. J. Roentgenology 342-44 (1950)).

148 See Henry Kempe et al., The Battered-Child Syndrome, 9 Child Abuse & Neglect 143, 144 (1985).

149 See id. at 143.

150 Id.

151 Id.

152 See Al-Holou et al., supra note 3, at 480.

153 Id. at 478-80 (citing A.N. Guthkelch, Infantile Subdural Haematoma and its Relationship to Whiplash Injuries, 2 Brit. Med. J. 430, 430-31 (1971); A.K. Ommaya et al., Whiplash Injury and Brain Damage: An Experimental Study, 204 JAMA 285, 285-89 (1968); A.K. Ommaya & A.E. Hirsch, Tolerances for Cerebral Concussion from Head Impact and Whiplash in Primates, 4 J. Biomechanics 13, 13-21 (1971); A.K. Ommaya & P. Yarnell, Subdural Haematoma After Whiplash Injury, 2 LANCET 237, 237-39 (1969)).

154 Id. at 480.

155 Id. (quoting A.N. Guthkelch, Infantile Subdural Hematoma and Its Relationship to Whiplash Injuries, 2 Brit. Med. J. 430, 430-31 (1971)).

156 Id. (citing J. Caffey, On the Theory and Practice of Shaking Infants. Its Potential Residual Effects of Permanent Brain Damage and Mental Retardation, 124 Am. J. Diseases Child. 161-69 (1972); J. Caffey, The Parent-Infant Traumatic Stress Syndrome; (Caffey-Kempe Syndrome), (Battered Babe Syndrome), 114 Am. J. Roentgenology Radium Therapy Nuclear Med. 218-29 (1972); J. Caffey, The Whiplash Shaken Infant Syndrome: Manual Shaking by the Extremities With Whiplash-Induced Intracranial and Intraocular Bleedings, Linked With Residual Permanent Brain Damage and Mental Retardation, 54 Pediatrics 396-403 (1974)).

157 See Kleinman & Barnes, supra note 141, at 298.

158 See Tuerkheimer, supra note 4, at 1, 12.

159 Id. at 11.

160 Id. at 12.

161 Id. at 12-13 (quoting Donohoe, Evidence-Based Medicine, supra note 12); see also Gena, supra note 4, at 710-14 (quoting Donohoe).

162 Id. at 14; see also Gena, supra note 4, at 710.

163 David H. Kaye & David A. Freedman, Reference Guide on Statistics, in Reference Manual on Scientific Evidence 83, 85 (2nd ed. 2000), http://www.fjc.gov/public/pdf.nsf/lookup/sciman02.pdf/$file/sciman02.pdf.

164 For a more detailed analysis of statistics and the law, see Panel on Statistical Assessments as Evidence in the Courts, National Research Council, The Evolving Role of Statistical Assessments as Evidence in the Courts (Stephen E. Fienberg ed., 1989); Michael O. Finkelstein & Bruce Levin, Statistics for Lawyers (2d ed. 2001).

165 Kaye & Freedman, supra note 163, at 90.

166 Id.

167 Id. at 91 ("'Anecdotal evidence' means reports of one kind of event after following another." But, such reports are often chosen "haphazardly or selectively," and do not "demonstrate that the first event causes the second.").

168 See id. at 90-91.

169 Id.; see also Haggerty v. Upjohn Co., 950 F. Supp. 1160, 1163-64 (S.D. Fla. 1996) (discussing the use of anecdotal case reports to generate hypotheses about causation).

170 Kaye & Freedman, supra note 163, at 91. As described below, observational studies are susceptible to "confounding variables" and bias. See id. at 92. Bias can take many forms (selection, observation, recall, and reporting, to name a few), and can affect both observational and experimental studies.

171 See id. at 91.

172 See Glossary, BMJ, http:// clinicalevidence.bmj.com/ceweb/resources/glossary.jsp (last visited Nov. 17, 2011) (defining "case control study" and "observational studies").

173 See id.

174 Kaye & Freedman, supra note 163, at 92.

175 Id.

176 Id.

177 Id.

178 Id. at 93.

179 Id. The analytical procedure most commonly used in statistics to control for confounding in observational studies is regression analysis. See id. at 94 n.31.

180 Id. at 94.

181 Id. at 95. For example, the evidence that smoking causes lung cancer is largely observational, but still very compelling. Id.

182 Id. at 94.

183 Id. at 96.

184 Id. at 96.

185 Id. at 102.

186 See generally Headache Classification Subcommittee of the International Headache Society (IHS), The International Classification of Headache Disorders (2d ed., 1st rev. 2005), http://216.25.88.43/upload/CT_ Clas/ICHD-IIR1final.pdf.

187 See id. at 28.

188 Whereas RCTs are not optimal for diagnostic studies, they are the study of choice for assessing therapies. See Jan P. Vandenbroucke, Observational Research, Randomised Trials, and Two Views of Medical Science, 5 PLoS Med. 0339, 0340 (2008) ("Randomised controlled trials are rarely used for research to detect or to establish causes of disease, mainly because randomisation is most of the time impossible, but quite fortunately, randomisation is most of the time not needed."); see also Alvan R. Feinstein & Ralph I. Horwitz, Problems in the "Evidence" of "Evidence-Based Medicine," 103 Am. J. Med. 529, 529 (1997) ("Randomized trial information is also seldom available for issues in etiology, diagnosis, and prognosis....").

189 See Tuerkheimer, supra note 4, at 12 nn.65, 67-70; Gena, supra note 4, at 706 n.56.

190 See generally Donohoe, supra note 12.

191 Id. at 239-40.

192 Id. at 240.

193 Id.

194 Id.

195 Id. at 241 (emphasis added); see also Tuerkheimer, supra note 4, at 12, 32.

196 See Donohoe, supra note 12, at 241.

197 Id.

198 See id. at 240.

199 In fact, in the article itself, the author admits missing what he himself considers an "important" study by Jayawant et al. using his own search criteria. See id. at 240.

200 See Donohoe, supra note 12, at 240-41.

201 Id. at 239-41.

202 See Cavazos v. Smith, 132 S. Ct. 2, 10 (2011) (per curiam) (Ginsburg, J., dissenting). See generally Tuerkheimer, supra note 4, at 12 & n.70 (citing evidentiary hearing testimony of Patrick Barnes in State v. Edmunds, 746 N.W.2d 590 (Wis. Ct. App. 2008)); Gena, supra note 4, at 727.

203 See Kaye & Freedman, supra note 163, at 115.

204 Id.

205 Id.

206 Id. at 116. While posterior probabilities, the applicability of the statistical models, and regression analysis are other important considerations, for the limited purposes of this article, we will focus on precision of data and statistical significance. For a more detailed discussion of the topic, I would guide the reader to Kaye & Freedman, supra note 163, at 116-78.

207 Id. at 117.

208 Id. at 115 n.107. The "mean" of data is the average of the data. Id. at 114 n.102.

209 Id. at 118.

210 Id. at 174.

211 Id.

212 See id. at 118, 174.

213 Id. at 118.

214 Id. at 118-19.

215 Id. at 118.

216 Id. at 119 (footnotes omitted).

217 Id. at 168.

218 See id. at 122.

219 See id. at 168. See also id. at 124 n.142 (quoting Waisome v. Port Auth. N.Y. & N.J., 948 F.2d 1370, 1376 (2d Cir. 1991) ("Social scientists consider a finding of two standard deviations significant, meaning there is about one chance in 20 that the explanation for a deviation could be random...."); Rivera v. City of Wichita Falls, 665 F.2d 531, 545 n.22 (5th Cir. 1982) ("A variation of two standard deviations would indicate that the probability of the observed outcome occurring purely by chance would be approximately five out of 100; that is, it could be said with a 95% certainty that the outcome was not merely a fluke.")).

220 Kay & Freedman, supra note 163, at 168. Computing the p-value requires statistical experience and is reserved for those with expertise in statistics and epidemiology. See id. at 87, 123. Incidentally, some statisticians point out that a determination of "statistical significance" is not as important as understanding how analysts developed their models. See id. at 128. For example:
If enough comparisons are made, random error almost guarantees that some will yield "significant" findings, even when there is no real effect. Consider the problem of deciding whether a coin is biased. The probability that a fair coin will produce ten heads when tossed ten times is $(1/2)^{10} = 1/1,024$. Observing ten heads in the first ten tosses, therefore, would be strong evidence that the coin is biased. Nevertheless, if a fair coin is tossed a few thousand times, it is likely that at least one string of ten consecutive heads will appear. Id. at 127; see also id. at 124, n.140; (citing John C. Bailar III & Frederick Mosteller, Guidelines for Statistical Reporting in Articles for Medical Journals: Amplifications and Explanations, in Medical Uses of Statistics, (2d ed. 1992) ("Merely labeling results as 'significant' or 'not significant' without providing the underlying information that goes into this conclusion is of limited value.").

221 Kay & Freedman, supra note 163, at 122.

222 Id. at 172.

223 See id.

224 Id. at 173. A test with high specificity for a condition will have a low rate of false positives. See id. at 172-73

225 See id. at 173.

226 Finkelstein & Levin, supra note 164, at 82.

227 See id.

228 Penny F. Whiting et al., Graphical Presentation of Diagnostic Information, BMC Med. Research Methodology, tbl.1 (Apr. 11 2008), http:// www.biomedcentral.com/content/pdf/1471-2288-8-20.pdf; see also, Finkelstein & Levin, supra note 164, at 83.

229 Stats: What is an Odds Ratio?, Children's Mercy, http:// www.childrens-mercy.org/stats/definitions/or.htm (last visited July 8, 2011).

230 Kaye & Freedman, supra note 163, at 167.

231 See id.

232 See Donohoe, supra note 12, at 241; see also Tuerkheimer, supra note 4; Gena, supra note 4 (authors who have just "reified" Donohoe's assertions).

233 This abbreviated bibliography is focused primarily on the literature in the past twelve years, as assertions have been made that there has been a "shifted consensus" in the medical community against the legitimacy of the Abusive Head Trauma diagnosis, which is predicated upon "new research." See Tuerkheimer, supra note 4, at 15-29.

234 For a completely comprehensive review of the topic, I would reference the reader to a review of the treatises on the topic (listed herein below in notes 235 & 236) as a starting point, with a subsequent search of the Medline database using broad search terms such as "subdural hemorrhage" or "retinal hemorrhage," with appropriately limiting criteria (i.e., including only children, excluding comments/editorials etc.). Assistance from a medical librarian may be required.

235 See Am. Acad. of Pediatrics, Inflicted Childhood Neurotrauma: Proceedings of a Conference Sponsored by Department of Health and Human Services, National Institute of Health, National Institute of Child Health and Human Development, Office of Rare Disease, and National Center for Medical Rehabilitation Research (Robert M. Reece & Carol E. Nicholson eds., 2003); Frasier et al., supra note 13.

236 See Child Abuse and Neglect: Diagnosis, Treatment, and Evidence 35-38, 347-457 (Carole Jenny ed., 2010) (chapters 6, 39-48); Suzanne Starling, Head Injury in Child Maltreatment: A Clinical Guide and Photographic Reference 37 (Angelo P. Giardino & Randell Alexander eds., 2003); Kleinman & Barnes, supra note 141; Rorke-Adams et al., supra note 13.

237 In coming to this safe estimation, this author conducted an all language literature search of the Medline database from 1970 to March 2010, using over 15 different keywords/phrases (to include, but not limited to, "shaken baby syndrome," "shaken infant syndrome," "inflicted neurotrauma," "nonaccidental trauma," "subdural hemorrhage," "subarachnoid hemorrhage," and "retinal hemorrhage"). All meta-analyses, practice guidelines, randomized control trials, case reports, comparative studies, controlled clinical trials, historical or classical articles, multicenter studies and technical reports in children under eighteen years of age were included. All reviews, comments, editorials, letters, and news articles were excluded. The restricted searches to the search terms "subdural hemorrhages" and "retinal hemorrhages" by themselves produced over 1000 abstracts and over 500 abstracts, respectively. This author then reviewed over 1000 abstracts from the above searches to gauge applicability to the topic of Abusive Head Trauma, and safely determined that at least 700 articles were pertinent to the topic. Additionally, given the non-comprehensive nature of the search (i.e., the limitation to one database and a non-exhaustive list of keywords/phrases), this author was able to safely conclude that the above-stated number of studies was an underestimate.

238 The different nationalities publishing on this topic include: Argentina, Australia, Belgium, Brazil, Canada, China, Czech Republic, Denmark, Estonia, Finland, France, Germany, Greece, India, Israel, Italy, Japan, Malaysia, Netherlands, New Zealand, Norway, Poland, Russia, Singapore, Spain, Switzerland, United Kingdom, and the United States.

239 See infra Appendix A.

240 See Donohoe, supra note 12, at 240-41.

241 See Rorke-Adams et al., supra note 13, at 61.

242 Id.

243 Id. at 64.

244 Id. at 65 tbl.2.2, 81-84.

245 Id. at 61, 63-64.

246 See, e.g., C. Hobbs et al., Subdural Haematoma and Effusion in Infancy: An Epidemiological Study, 90 Archives Disease Childhood 952, 954.

247 Id. at 952-53.

248 Id. at 953 tbl.2. "Underdetermined cause" combines Hobbs' "Perinatal" and "Undetermined" categories, and "Traumatic SDHs" combines Hobbs' "Accident" and "Abuse" categories. See id.

249 Id.

250 See S. Jayawant et al., Subdural Haemorrhages in Infants: Population Based Study, 317 Brit. Med. J. 1558, 1559, 1561 (1998); Victoria Trenchs et al., Subdural Haematomas and Physical Abuse in the First Two Years of Life, 43 Pediatric Neurosurgery 352, 352-53, 356 (2007); Dimitra Tzioumi & R. Kim Oates, Subdural Hematomas in Children Under 2 Years. Accidental or Inflicted? A 10-Year Experience, 22 Child Abuse & Neglect 1105, 1106-07 (1998).

251 Kenneth W. Feldman et al., The Cause of Infant and Toddler Subdural Hemorrhage: A Prospective Study, 108 Pediatrics 636, 638 (2001) (source also located in Appendix A, "General" literature, prospective article #14).

252 Id. at 637.

253 Id. at 637-38.

254 Id. at 638 tbl.2.

255 E.g., Jakob Matschke et al., Nonaccidental Head Injury is the Most Common Cause of Subdural Bleeding in Infants < 1 Year of Age, 124 Pediatrics 1587, 1594 (2009) (source also located in Appendix A, "Pathology" literature, retrospective article #20).

256 Id. at 1588.

257 Id. at 1589.

258 Id.

259 Id.

260 Id. at 1594.

261 As discussed in the statistics section above, the term "specific" in this context is used with regards to its statistical definition; meaning that it is a condition/injury that can produce some false positives with regards to AHT. See Kaye & Freedman, supra note 163, at 173 (definition of specificity).

262 E.g., A.C. Duhaime et al., Head Injury in Very Young Children: Mechanisms, Injury Types, and Ophthalmologic Findings in 100 Hospitalized Patients Younger than 2 Years of Age, 90 Pediatrics 179, 183 (1992) (source also located in Appendix A, "General" literature, prospective article #10).

263 Id. at 179, 181.

264 Id. at 179.

265 See id. at 179-80, 184.

266 Id. at 180.

267 See id. at 181

268 Id. at 183.

269 See id. at 184. Recall the general statistical principles section above: p-value is essentially the likelihood the result is due to chance.

270 Id. at 181, 184.

271 See Kirsten Bechtel, et al., Characteristics that Distinguish Accidental from Abusive Injury in Hospitalized Young Children with Head Trauma, 114 Pediatrics 165, 165, 168 (2004) (source also located in Appendix A, "General" literature, prospective article #5).

272 Id. at 166.

273 Id. at 166 tbl.1.

274 See id.

275 Id. at 166.

276 Id. at 167, tbl.3.

277 Id.

278 See id.

279 See id.

280 See Matthieu Vinchon et al., Confessed Abuse Versus Witnessed Accidents in Infants: Comparison of Clinical, Radiological, and Ophthalmological Data in Corroborated Cases, 26 Child's Nervous Sys. 637, 638-39 (2010) (source also located in Appendix A, "General" literature, prospective article #23).

281 Id. Confessions were obtained from judicial sources. Id. at 638.

282 Id. at 641 tbl.2.

283 Id.

284 See id. at 639, 641 tbl.2.

285 See Linda Ewing-Cobbs, et al., Neuroimaging, Physical, and Developmental Findings after Inflicted and Noninflicted Traumatic Brain Injury in Young Children, 102 Pediatrics 300, 300 (1998); Carla DiScala, et al., Child Abuse and Unintentional Injuries, 154 Archives Pediatrics & Adolescent Med. 16, 16 (2000); Kent P. Hymel et al., Head Injury Depth as an Indicator of Causes and Mechanisms, 125 Pediatrics 712, 715-18 (2010) [hereinafter Hymel et al., Head Injury Depth]; Kent P. Hymel et al., Mechanisms, Clinical Presentations, Injuries, and Outcomes from Inflicted Versus Noninflicted Head Trauma during Infancy: Results of a Prospective, Multicentered, Comparative Study, 119 Pediatrics 922, 922 (2007) [hereinafter Hymel et al., Mechanisms]; Heather T. Keenan et al., A Population-Based Comparison of Clinical Outcome Characteristics of Young Children with Serious Inflicted and Noninflicted Traumatic Brain Injury, 114 Pediatrics 633, 633 (2004); Mark W. Morris et al., Evaluation of Infants with Subdural Hematoma who Lack External Evidence of Abuse, 105 Pediatrics 549, 549 (2000); M.C. Myhre et al., Traumatic Head Injury in Infants and Toddlers, 96 Acta Paediatrica 1159, 1159 (2007); Robert M. Reece & Robert Sege, Childhood Head Injuries, 154 Archives Pediatrics & Adolescent Med. 11, 11 (2000); Shervin R. Dashti et al., Current Patterns of Inflicted Head Injury in Children, 31 Pediatric Neurosurgery 302, 302 (1999); Matthieu Vinchon et al., Accidental and Nonaccidental Head Injuries in Infants: A Prospective Study, 102 J. Neurosurgery: Pediatrics 380, 380-81 (2005) (sources also referenced in Appendix A, "General" literature, prospective articles #12, 18, 19 & 21; retrospective articles # 12, 27, 28, & 32; "Neurosurgery" literature, prospective article #3 and retrospective article #5).

286 Robert G. Wells et al., Intracranial Hemorrhage in Children Younger than 3 Years, 156 Archives Pediatrics & Adolescent Med. 252, 253, 254 tbl.2 (2002) (source also referenced in Appendix A, "Radiology" literature, retrospective article #14).

287 Id. at 253.

288 Id.

289 Id.

290 Id.

291 Id. at 254 tbls.1 & 2.

292 Id. at 255.

293 See id. at 255 & tbl.3.

294 See S. Datta et al., Neuroradiological Aspects of Subdural Haemorrhages, Archives Disease Childhood 947, 948, 950 (2005); Hymel et al., Mechanisms, supra note 285, at 928. But see Glenn A. Tung et al., Comparison of Accidental and Nonaccidental Traumatic Head Injury in Children on Noncontrast Computed Tomography, 118 Pediatrics 626, 632 (2006) (showing authors did not find a significant statistical association with interhemispheric SDHs and non-accidental trauma) (source also referenced in Appendix A, "Radiology" literature, comparative article #8).

295 Datta et al., supra note 294, at 947-48.

296 Alex V. Levin, Retinal Hemorrhages: Advances in Understanding, 56 Pediatric Clinics N. Am. 333, 335 (2009) (source also referenced in Appendix A, "Ophthalmology" literature, review article #5).

297 Id.

298 Id.

299 Id.

300 Id.

301 See id. at 338.

302 Id. at 335.

303 Id. at 333, 341.

304 Id. at 335.

305 Id.

306 Id.

307 Id.

308 Id. at 335-36; see Gregg T. Lueder et al., Perimacular Retinal Folds Simulating Nonaccidental Injury in an Infant, 124 Archives Ophthalmology 1782, 1782-83 (2006) (source also referenced in Appendix A, "Ophthalmology" literature, retrospective article #22); P.E. Lantz et al., Perimacular Retinal Folds from Childhood Head Trauma, 328 Brit Med. J. 754, 754 (2004) (source also referenced in Appendix A, "Pathology" literature, retrospective article #16); Ajay Bhatnagar et al., Subinternal Limiting Membrane Hemorrhage with Perimacular Fold in Leukemia, 127 Archives Ophthalmology 1548, 1548 (2009) (source also referenced in Appendix A, "Ophthalmology" literature, retrospective article #4); JD Kivlin et al., Retinal Hemorrhages in Children Following Fatal Motor Vehicle Crashes: A Case Series, 126 Archives Ophthalmology 800, 800-01 (2008) (source also referenced in Appendix A, "Ophthalmology" literature, retrospective article #18).

309 See, Levin, supra note 296, at 334 box1.

310 Id. at 333, 335.

311 See Levin, supra note 296, at 337.

312 See M. Vaughn Emerson et al., Incidence and Rate of Disappearance of Retinal Hemorrhage in Newborns, 108 Ophthalmology 36, 36 (2001); Lindsey A. Hughes et al., Incidence, Distribution, and Duration of Birth-Related Retinal Hemorrhages: A Prospective Study, 10 J. Am. Ass'n for Pediatric Ophthalmology & Strabismus 102, 102 (2006) (sources also referenced in Appendix A, "Ophthalmology" literature, prospective articles #6 & #13).

313 See Emerson et al., supra note 312, at 36.

314 Id. at 37.

315 Id. at 38. There are rare cases of birth-related RHs lasting until six to eight weeks of life. See id. There has been no documentation of birth related RHs outside of eight weeks (two months) of life. See id.; Hughes et al., supra note 312, at 106.

316 Id. at 39.

317 See Levin, supra note 296, at 334 box1.

318 Id. at 337.

319 Id.

320 See James C. Fackler et al., Retinal Hemorrhages in Newborn Piglets Following Cardiopulmonary Resuscitation, 146 Am. J. Diseases Children 1294, 1295 (1992); M.G.F. Gilliland & Martha Waters Luckenbach, Are Retinal Hemorrhages Found After Resuscitation Attempts? A Study of the Eyes of 169 Children, 14 Am. J. Forensic Med. & Pathology 187, 189 (1993); Amy Odom et al., Prevalence of Retinal Hemorrhages in Pediatric Patients After In-hospital Cardiopulmonary Resuscitation: A Prospective Study, 99 Pediatrics, at *4 (June 2007) (sources also referenced in Appendix A, "Ophthalmology" literature, controlled study #2 & prospective articles #7 & #16).

321 See Odom et al., supra note 320, at *2.

322 Id.

323 Id. at *4.

324 Id. at *1.

325 Id. at *1, *4.

326 See id. at *3-*4.

327 See A.I. Curcoy et al., Do Retinal Haemorrhages Occur in Infants with Convulsions?, 94 Archives Disease Childhood 873, 874 (2009) (seizures); Michael Goldman et al., Severe Cough and Retinal Hemorrhage in Infants and Young Children, 148 J. Pediatrics 835, 836 (2006) (coughing); Sandra Herr et al., Does Valsalva Retinopathy Occur in Infants? An Initial Investigation in Infants with Vomiting Caused by Pyloric Stenosis, 113 Pediatrics 1658, 1660 (2004) (vomiting); M. Mei-Zahav et al., Convulsions and Retinal Haemorrhage: Should We Look Further? 86 Archives Disease Childhood 334, 334-35 (2002) (convulsions); S. Sandramouli et al., Retinal Hemorrhages and Convulsions, 76 Archives Disease Childhood 449, 449-50 (1997) (seizures) Ajai K. Tyagi et al., Can Convulsions Alone Cause Retinal Haemorrhages in Infants? 82 Brit. J. Ophthalmology 659, 659-60 (1998) (seizures); (sources also referenced in Appendix A, "Ophthalmology" literature, prospective articles #5, 11, 12, 15, 19, & 23). One other mechanism of retinal hemorrhaging occasionally mentioned is Purtscher's Syndrome. Levin, supra note 296, at 337. Purtscher's syndrome is the presence of certain characteristically-patterned RHs (hexagonal with white patches) that occur in adults that suffer severe crush chest injury. Id. The particular characteristically-patterned RHs (Purtscher's retinopathy) are rarely seen in AHT and are most likely the result of infarction, fat emboli from broken bones, or inflammation-mediated change. Id.

328 See Susan Schloff et al., Retinal Findings in Children with Intracranial Hemorrhage, 109 Ophthalmology 1472, 1472 (2002) (source also referenced in Appendix A, "Ophthalmology" literature, prospective article #20).

329 Id. at 1473.

330 Id. at 1473 tbl.1.

331 Id. at 1473.

332 Id. at 1472.

333 Id.

334 Id. at 1473-74.

335 See Yair Morad et al., Correlation Between Retinal Abnormalities and Intracranial Abnormalities in the Shaken Baby Syndrome, 134 Am. J. Ophthalmology 354, 355-56 (2002); (source also referenced in Appendix A, "Ophthalmology" literature, retrospective article #30).

336 See Levin, supra note 296, at 338.

337 See Yvonne M. Buys et al., Retinal Findings After Head Trauma in Infants and Young Children, 99 Ophthalmology 1718, 1720 (1992); Cindy W. Christian et al., Retinal Hemorrhages Caused by Accidental Household Trauma, 135 J. Pediatrics 125, 127 (1999); Dennis L. Johnson et al., Accidental Head Trauma and Retinal Hemorrhage, 33 Neurosurgery 231, 231-32 (1993); V. Trenchs et al., Retinal Haemorrhages in Head Trauma Resulting from Falls: Differential Diagnosis with Non-Accidental Trauma in Patients Younger than 2 Years of Age, 24 Child's Nervous Sys. 815, 817 (2008); V. Sturm et al., Rare Retinal Haemorrhages in Translational Accidental Head Trauma in Children, 23 Eye 1535, 1540 (2009); Kivlin et al., supra note 308, at 803 (sources also referenced in Appendix A, "Ophthalmology" literature, prospective articles #3, 14, & 22; retrospective articles #8, 18, & 36). In the rare instances when RHs were present, there were only a few preretinal or intraretinal RHs confined to the posterior pole. See, e.g., Cindy W. Christian et al., Retinal Hemorrhages Caused by Accidental Household Trauma, 135 J. Pediatrics 125, 125-27 (1999).

338 See Levin, supra note 296, at 338.

339 Id.

340 Id.

341 Id.

342 See id. at 341.

343 See Vinchon et al., supra note 285, at 380.

344 Id. 380-81.

345 Id. at 381.

346 Id.

347 Id. at 382.

348 Id.

349 See Vinchon et al., supra note 280, at 637-38, 644.

350 See id. at 637-38.

351 Id. at 641 tbl.2.

352 Id.

353 Id.

354 Id. at 640 tbl.1.

355 Id. at 642 tbl.4.

356 Id. at 642 tbl.4, 643.

357 Id. at 644.

358 See Vincent Pierre-Kahn et al., Ophthalmologic Findings in Suspected Child Abuse Victims with Subdural Hematomas, 110 Ophthalmology 1718, 1720 (2003) (source also referenced in Appendix A, "Ophthalmology" literature, prospective article #17); Bechtel et al., supra note 271, 166-67; Reece & Sege, supra note 285, at 13-14.

359 See Gaurav Bhardwaj et al., A Systematic Review of the Diagnostic Accuracy of Ocular Signs in Pediatric Abusive Head Trauma, 117 Ophthalmology 983, 987 tbl.1 (2010) (presenting results of Riffenburgh study) (Bhardwaj source also referenced in Appendix A, "Ophthalmology" literature, systematic review #2; Riffenburgh source also referenced in Appendix A, "Pathology" literature, controlled study #7).

360 Id.

361 Id.

362 See id.; infra Part II.B.1.b.

363 See, e.g., Aaron M. Gleckman et al., Optic Nerve Damage in Shaken Baby Syndrome, 124 Archives Pathology & Laboratory Med. 251, 252 tbl., 255 (2000) (source also referenced in Appendix A, "Pathology" literature, controlled study #4); Donald L. Budenz et al., Ocular and Optic Nerve Hemorrhages in Abused Infants with Intracranial Injuries, 101 Ophthalmology 559, 561 (1994) (source also referenced in Appendix A, "Pathology" literature, controlled study #2); Gilliland & Luckenbach, supra note 320, at 191.

364 See S. Maguire, Which Clinical Features Distinguish Inflicted from Non-Inflicted Brain Injury? A Systematic Review, 94 Archives Disease Childhood 860, 860 (2009) (source also referenced in Appendix A, "General" literature, systematic review article #4).

365 Id. at 861, 864 fig.1.

366 Id. at 863-64.

367 Id. at 861.

368 Id.

369 Id. at 865.

370 Id.; see Maguire et al., supra note 364, at 865.

371 Bhardwaj et al., supra not 359, at 984.

372 Id.

373 Id.

374 Bhardwaj, supra note 359, at 991. "Level I evidence provides strong support for a statement, and is usually composed of well-performed, randomized controlled-trials or meta-analyses of randomized controlled-trials. Level II evidence provides substantial support for the statement... [and] usually includes observational studies, such as cohort studies and case control studies. Level III indicates a weak body of evidence relying on consensus statements, small noncomparative case series, and individual case reports." Id. at 984; see also Alex V. Levin et al., Clinical Report: The Eye Examination in the Evaluation of Child Abuse, 126 Pediatrics 376, 376-77 (2010) (discussing use of intraocular hemorrhage diagnoses in assessing AHT) (source also referenced in Appendix A, "Ophthalmology" literature, systematic review #4).

375 See Bhardwaj, supra note 359, at 990-91.

376 See J. Haviland & R.I. Ross Russell, Outcome After Severe Non-Accidental Head Injury, 77 Archives Disease Childhood 504, 504-05 (1997) (source also found in Appendix A, "General" literature, comparative study #16).

377 Id. at 505.

378 Id.

379 Id. At discharge nine AHT survivors were deemed severe, three fell in the moderate category, and one patient was normal. Id. at 506 tbl.3.

380 Id. at 505.

381 Id. at 505, 506 tbl.4. The article presents conflicting data. The body of the article only accounts for eight of the nine survivors, stating six of the survivors were deemed normal at discharge. Id. at 505. Because the percentages stated on page 505 do not add up 100%, I relied on Table 4 data, which showed seven survivors had a normal status at discharge. See id. at 505, 506 tbl.4.

382 See id.

383 See Heather T. Keenan et al., Neurodevelopmental Consequences of Early Traumatic Brain Injury in 3-Year-Old Children, 119 Pediatrics e616, e619-e620 (2007) (source also referenced in Appendix A, "General" literature, controlled study #5); Matthieu Vinchon et al., Infantile Traumatic Subdural Hematomas: Outcome after Five Years, 39 Pediatric Neurosurgery 122, 124-25 (2003) (source also referenced in Appendix A, "Neurosurgery" literature, prospective study #4); Linda Ewing-Cobbs et al., Late Intellectual and Academic Outcomes Following Traumatic Brain Injury Sustained During Early Childhood, 105 J. Neurosurgery: Pediatrics 287 (2006), http:// www.ncbi.nlm.nih.gov/pmc/articles/PMC2615233/pdf/nihms23194.pdf (source also referenced in Appendix A, "General" literature, comparative studies #11); Hymel et al., Mechanisms, supra note 285, at 924-25, 927 tbl.4; Vinchon et al., supra note 280, at 641 tbl.3; Ewing-Cobbs et al., supra note 285, at 303-04.

384 Joeli Hettler & David S. Greenes, Can the Initial History Predict Whether a Child with a Head Injury has been Abused?, 111 Pediatrics 602, 602 (2003) (source also referenced in Appendix A, "General" literature, retrospective article #17).

385 Id.

386 Id.

387 Id. at 603.

388 Id. at 602, 605 tbl.4.

389 Id. at 604.

390 Duhaime et al., supra note 262, at 184; see Heather T. Keenan et al., Child Outcomes and Family Characteristics 1 Year After Severe Inflicted or Noninflicted Traumatic Brain Injury,117 Pediatrics 317, 317 (2006); see also Keenan et al., supra note 285, at 637.

391 See Tuerkheimer, supra note 4, at 13 & n.76 (citing the testimony of a defense expert, neuroradiologist Dr. Patrick Barnes, in State v. Edmunds). The assertion is that writers of much of the medical literature on Abusive Head Trauma "select[ed] cases by the presence of the very clinical findings and test results they [sought] to validate as diagnostic." Id. at 13 As Dr. Barnes simply stated, "SBS=SDH + RH [inclusion criteria], therefore, SDH + RH=SBS [conclusion]." Id.

392 See supra Section II.A. (explaining the direct contributions of these and other authors).

393 Id.

394 Id. (detailing studies that accounted for "circularity").

395 There have been a few other hypothesized mechanisms (such as increased intrathoracic pressure) for SDHs. But addressing all of these hypothesized mechanisms is beyond the scope of this article. For further information regarding these hypothesized mechanisms, I would refer the reader to Frasier et al, supra note 14, and Rorke-Adams et al., supra note 14.

396 J.F. Geddes et al., Dural Haemorrhage in Non-Traumatic Infant Deaths: Does it Explain the Bleeding in "Shaken Baby Syndrome"?, 29 Neuropathology & Applied Neurobiology 14, 14 (2003) (source also referenced in Appendix A, "Pathology" literature, retrospective article #9); Waney Squier & Julie Mack, The Neuropathology of Infant Subdural Haemorrhage, 187 Forensic Sci. Int'l 6, 12 (2009); Julie Mack et al., Anatomy and Development of the Meninges: Implications for Subdural Collections and CSF Circulation, 39 Pediatric Radiology 200, 200 (2009) (sources also referenced in Appendix A, "Pathology" literature, review articles #6 & 9).

397 Geddes et al, supra note 396, at 14 (emphasis added).

398 See id. at 19.

399 Id. at 15.

400 Id.

401 See id. at 15, 17 tbl.2.

402 Id. at 15.

403 See generally id.

404 See id. at 15, 19.

405 See id. at 17.

406 Id.

407 See id.

408 See id.

409 See supra Section II.B.2.a.

410 See Marta C. Cohen & Irene Scheimberg, Evidence of Occurrence of Intradural and Subdural Hemorrhage in the Perinatal and Neonatal Period in the Context of Hypoxic Ischemic Encephalopathy: An Observational Study from Two Referral Institutions in the United Kingdom, 12 Pediatric & Developmental Pathology 169 (2009); (source also referenced at Appendix A, "Pathology" literature, prospective article #1); see also infra Part III.A (discussing the legal ramifications of the "Unified Hypothesis" in United Kingdom courts).

411 Cohen & Scheimberg, supra note 410, at 169.

412 Id.

413 See generally id.

414 See C. Smith, & J. Bell, Shaken Baby Syndrome: Evidence and Experts, 50 Dev. Med. Child Neurology 6, 6 (2008).

415 See generally Cohen & Scheimberg, supra note 410.

416 See generally id.

417 Compare Geddes et al., supra note 396, at 14 (using fetuses with gestational ages of 18 to 41 weeks and newborn with ages as high as five months) with Cohen & Scheimberg, supra note 410, at 169 (using fetuses with gestational ages of 26 to 40 weeks and newborns with ages between 1 hour and 19 days).

418 Geddes et al., supra note 396, at 14.

419 Tim Jaspan, Current Controversies in the Interpretation of Non-Accidental Head Injury, 38 Pediatric Radiology s378, s382 (Supp. 2008) (source also referenced in Appendix A, "Radiology" literature, review #3).

420 See generally Benjamin Y. Huang & Mauricio Castillo, Hypoxic-Ischemic Brain Injury: Imaging Findings from Birth to Adulthood, 28 Radiographics 417, 433 (2008).

421 See Jaspan, supra note 419, at s382.

422 See David J. Dubowitz et al., MR of Hypoxic Encephalopathy in Children after Near Drowning: Correlation with Quantitative Proton MR Spectroscopy and Clinical Outcome, 19 Am J. Neuroradiology 1617, 1618 (1998) (source also referenced in Appendix A, "Radiology" literature, retrospective article #7).

423 Id. at 1620-22, 1626.

424 See O. Baenziger et. al., Early Pattern Recognition in Severe Perinatal Asphyxia: A Prospective MRI Study, 35 Neuroradiology 437, 440 (1993); A. James Barkovich et. al., Perinatal Asphyxia: MR Findings in the First 10 Days, 16 Am. J. Neuroradiology 427, 427 (1995); Mary Rutherford et al., Hypoxic-ischaemic Encephalopathy: Early and Late Magnetic Resonance Imaging Findings in Relation to Outcome, 75 Archives Disease Childhood F145, F145, F151 (1996); L.T. Sie et al., MR Patterns of Hypoxic-Ischemic Brain Damage After Prenatal, Perinatal or Postnatal Asphyxia, 31 Neuropediatrics 128, 128 (2000).

425 See Karim T. Rafaat et al., Cranial Computed Tomographic Findings in a Large Group of Children with Drowning: Diagnostic, Prognostic, and Forensic Implications, 6 Pediatric Critical Care Med. 567, 567 (2008) (source also referenced in Appendix A, "Radiology" literature, retrospective article #11).

426 Id.

427 Id. at 567-68.

428 Jaspan, supra note 419, at s382.

429 Roger W. Byard et al., Lack of Evidence for a Causal Relationship Between Hypoxic-Ischemic Encephalopathy and Subdural Hemorrhage in Fetal Life, Infancy, and Early Childhood, 10 Pediatric & Developmental Pathology 348, 348 (2007) (source also referenced in Appendix A, "Pathology," literature, retrospective article # 2).

430 Id.

431 Id.

432 Id.

433 Id.

434 Id.

435 See M. Hurley, et al., Is There a Causal Relationship Between the Hypoxia-Ischaemia Associated with Cardiorespiratory Arrest and Subdural Haematomas? An Observational Study, 83 Brit. J. Radiology 736, 736-37 (2010) (source also referenced in Appendix A, "Pathology" literature, retrospective article #15).

436 Id. at 736.

437 Id.

438 Id. at 736-37.

439 Id. at 737.

440 Id.

441 Id. at 738.

442 Id.

443 Id.

444 See id. at 737.

445 Id. at 743 (emphasis added).

446 Matschke et al., supra note 255, at 1594.

447 See Squier & Mack, supra note 396, at 8.

448 Id. at 8-9.

449 Id. at 10.

450 See id. at 10, 12.

451 Much has recently been made of the biomechanical research arguing against the validity of AHT/SBS. See Bazelon, supra note 5. Given the ethical limitations of research in the field, much prior pediatric biomechanical research was scaled data based upon adult values. See Jason F. Luck et al., Tensile Mechanical Properties of the Perinatal and Pediatric PMHS Osteoligamentous Cervical Spine, 52 Stapp Car Crash J. 107, 107-09 (2008). This left what was described by many learned researchers in the field as a "significant void in pediatric cervical spine biomechanics." Id. at 107. Although recent biomechanical research upon post-mortem infants is an improvement on that prior data, it is still limited and approximate. See id. at 109. In fact, Luck et al. found that "juvenile animal surrogates estimate the stiffness of the human cervical spine fairly well." Id. at 107. Along that vein of approximate data, recent animal studies (specifically, seven to ten day-old anesthetized lambs vigorously shaken by humans) have produced the exact same injuries commonly found in AHT/SBS--subdural hemorrhages and retinal hemorrhages. See John W. Finnie et al., Diffuse Neuronal Perikaryal Amyloid Precursor Protein Immunoreactivity in an Ovine Model of Non-Accidental Head Injury (the Shaken Baby Syndrome), 17 J. Clinical Neuroscience 237, 237-39 (2010).

452 See, e.g., Brian J. Forbes et. al., Inflicted Childhood Neurotrauma (Shaken Baby Syndrome): Ophthalmic Findings, 41 J. Pediatric Ophthalmology & Strabismus 80, 86 (2004).

453 See Chiesa & Duhaime, supra note 13, at 317.

454 "Diffuse Axonal Injury" refers to damage of the brain to a widespread, not focal, area; it most commonly manifests as lesions of the white matter tracts of the brain. See Douglas H. Smith et al., Diffuse Axonal Injury in Head Trauma, 18 J. Head Trauma Rehabilitation 307, 308 (2003).

455 See Tuerkheimer, supra note 4, at 4 & n.18, 7 n.39.

456 Although not discussed in this review, "encephalopathy" is also associated with trauma.

457 See Kempe et al., supra note 148, at 143.

458 See Chiesa & Duhaime, supra note 13, at 321.

459 Id. at 319-20.

460 Id at 319.

461 Id. at 320. In certain cases, specifically, in certain cases of fatal AHT, a detailed physical examination either is impractical (secondary to the critical care needs of the child) or unwarranted, as further physical examination information will be obtained via autopsy. See id. at 323.

462 Id. at 320.

463 See Carole Jenny et al., Analysis of Missed Cases of Abusive Head Trauma, 282 JAMA 621, 623 & tbl.2 (1999) (showing physicians failed to detect AHT 31.2% of the time) (source also referenced in Appendix A, "General" literature, retrospective study #19); see also Hymel et al., Head Injury Depth, supra note 285, at 712, 716 tbl.3 (showing abused children might not show scalp or skull injury, but still may have brain injury).

464 See Chiesa & Duhaime, supra note 13, at 321 (discussing differential diagnoses)

465 See id.

466 See infra notes 523-24.

467 See Chiesa & Duhaime, supra note 13, at 320. In fact, a multidisciplinary child protection team approach has become the standard of care in many jurisdictions. See id. at 319.

468 See id. at 321.

469 See id. at 322.

470 See id. at 319-20.

471 Id. at 321.

472 See id. at 321, 323

473 See id. at 322.

474 This presumes that after reasonable medical investigation there is still no other discernible medical cause for the injuries.

475 See Kempe et al., supra note 148, at 143.

476 Tuerkheimer, supra note 4, at 5 (citing State v. Edmunds 746 N.W.2d 590, 598-99 (Wis. Ct. App. 2008)).

477 See Tuerkheimer, supra note 4, at 14 (emphasis added).

478 Some of the below listed organizations have explicitly acknowledged support through practice guidelines or similar promulgations, while others have implicitly done so by providing clinician or patient education materials on their websites.

479 See Jonathan Dart & Sarah Cumberland, Fragile Brain, Handle with Care, 87 Bull. World Health Org. 331, 331-32 (2009); Fact Sheet No. 150, Child Maltreatment, World Health Org. (Aug. 2010), http:// www.who.int/mediacentre/factsheets/fs150/en/index.html.

480 The Royal Coll. of Paediatrics & Child Health & Royal Coll. of Radiologists, Standards for Radiological Investigations of Suspected Non-Accidental Injury 10 (March 2008), http:// www.rcpch.ac.uk/sites/default/files/asset_ library/Publications/S/StandardsforRadiologicalInvestigationsD..

481 See id.

482 See G. Adams et al., Update from the Ophthalmology Child Abuse Working Party: Royal College Ophthalmologists, 18 Eye 795, 795-96 (2004) available at www.rcophth.ac.uk/page.asp?section=493&search=.

483 See Joint Statement on Shaken Baby Syndrome, Canadian Paediatric Soc'y, http://www.cps.ca/english/statements/pp/cps01-01.htm (last visited Oct. 23, 2011).

484 Christian et al., supra note 6, at 1410.

485 Alex V. Levin et al., Information Statement: Abusive Head Trauma/Shaken Baby Syndrome, Am. Acad. of Ophthalmology (June 2010), http:// one.aao.org/ce/practiceguidelines/clinicalstatements_content.aspx?cid=914163d5-5313-4c23-80f1-07167ee62579.

486 Info for Patients: Shaken Baby Syndrome, Am. Ass'n for Pediatric Ophthalmology & Strabismus, http://www.aapos.org/terms/conditions/97 (last visited Oct. 23, 2011).

487 See James S. Meyer, et al., ACR Appropriateness Criteria: Suspected Physical Abuse--Child, Am. Coll. Radiology http:// www.acr.org/SecondaryMainMenuCategories/quality_safety/app_ criteria/pdf/ExpertPanelonPediatricImaging/SuspectedPhysicalAbuseChildDoc9.aspx (last reviewed 2009).

488 See Liz Horsley, AAP Guidelines on Evaluating Suspected Child Physical Abuse, 77 Am. Fam. Physicians 1461, 1461-64 (2008), available at http://www.aafp.org/afp/2008/0515/p1461.html.

489 See Patient Education, Am. Coll. of Surgeons, http:// www.facs.org/patienteducation/patient-resources/nervoussystem.html (last visited Aug. 26, 2011).

490 Patient Information: Shaken Baby Syndrome, Am. Ass'n of Neurological Surgeons (Nov. 2005) http://www.aans.org/Patient%20Information.aspx (follow "Click here to view Conditions and Treatments" hyperlink; then follow "Shaken Baby Syndrome" hyperlink).

491 See Child Abuse, Pediatric Orthopaedic Soc'y of N. Am, http:// www.posna.org/education/StudyGuide/childAbuse.asp (last visited Oct. 23, 2011); Fractures Associated with Head Injury, Pediatric Orthopaedic Soc'y of N. Am., http:// www.posna.org/education/StudyGuide/fracturesAssociatedwithHeadInjury.asp (last visited Oct. 23, 2011)

492 See Doraliz Hidalgo & Bernard L. Lopez, Head Trauma in Children Younger Than 2 Years, Critical Decisions Emergency Med., Apr. 2007, at 16 (presenting instruction for emergency physicians).

493 Shaken Baby Syndrome, Am. Acad. of Neurology, http:// www.aan.com/apps/disorders/index.cfm?event=database%3adisorder.view&disorder_ id=1060 (last visited Oct. 23, 2011).

494 Daubert v. Merrell Dow Pharm., Inc., 509 U.S. 579, 592 (1993) (footnotes omitted).

495 Id. at 593.

496 Id.

497 Id. at 594.

498 Id. Other factors for a trial court's consideration include whether "the expert's qualifications are sufficient... [whether] the method has been put to non-judicial uses... 'whether the expert's proposed testimony grows naturally and directly out of research the expert has conducted independent of the litigation'... 'whether the expert has unjustifiably extrapolated from accepted premise to unfounded conclusion'... [and] 'whether the expert has adequately accounted for alternative explanations."' David v. Black & Decker (US) Inc., 629 F. Supp. 2d 511, 514 (W.D. Pa. 2009) (citing Magistrini v. One Hour Martinizing Dry Cleaning, 180 F. Supp. 2d 584, 594 (D.N.J. 2002), aff'd 68 Fed. Appx. 356 (3d Cir. 2003)) (citation omitted).

499 See supra Section II.B.2 ("Statistical Evidence").

500 See, e.g., Hymel et al., Head Injury Depth, supra note 285, at 712-13.

501 See, e.g., Dubowitz et al., supra note 422, at 1617 (using MRI in near drowning episodes); Wells et al., supra note 286, at 252 (assessment using CT).

502 See, e.g., Luck et al., supra note 451, at 107, 109 (showing use of a physical forces perspective).

503 See, e.g., Geddes et al., supra note 396, at 18-19.

504 See supra text accompanying notes 234-35.

505 See infra Appendix A.

506 See In re Neurontin Mktg., Sales Practices & Prod. Liab., 612 F.Supp. 2d 116, 140 (D. Mass. 2009) ("Statistical evidence significance is one of the factors the Court should examine when determining whether a drug can cause an adverse event."); see also Daubert v. Merrell Dow Pharm., Inc., 509 U.S. 579, 594 (1993) ("[I]n the case of a particular scientific technique, the court ordinarily should consider the known or potential rate of error.")

507 As stated in the general statistics section above, in social sciences and medicine, this "observed significance level" (the p-value) is usually set at 5% (or 0.05) for "statistically significant," or 1% (or 0.01) for "moderately high" statistical significance, and 0.1% (or 0.001) for "high or strong" statistical significance. See supra Section II(B)(1)(b).

508 See S. Maguire et al., supra note 364, at 860 (systematic review showing positive predictive value for RH of 71%); Vinchon et al., supra note 285, at 380 (recent study showing specificity of 93.2% for RH in AHT and 100% of severe RH in AHT); see also Vinchon et al., supra note 280, at 642 tbl.4 (recent study showing severe RH specificity of 0.974 and a positive predictive value of 0.961).

509 See Vinchon et al., supra note 280, at 637.

510 In the 1950s, two eminent psychologists, Campbell and Fiske, sought to provide validation for psychological assessment tools that assessed vague variables such as courteousness, honesty, self-centeredness, imaginativeness, talkativeness, etc. See Donald T. Campbell & Donald W. Fiske, Convergent and Discriminant Validation by the Multitrait-Multimethod Matrix, 56 Psychol. Bull. 81, 98 tbl.13 (1959). In creating the multitrait-multimethod approach to assessing validity of psychological assessment tools, Campbell and Fiske determined that one of the key components was the concept of "convergent validation." Id. at 81.

511 Id. at 81.

512 Id.

513 See People v. Martinez, 74 P.3d 316, 323 (Colo. 2003) ("[W]e assume, as it is not in dispute, that the scientific principles of shaken-impact syndrome and subdural hematomas resulting from extreme accidents are reasonably reliable"); State v. McClary, 541 A.2d 96, 102 (Conn. 1988) (shaken baby syndrome is generally accepted by medical science); State v. Torres, 121 P.3d 429, 437 (Kan. 2005) (testimony by physicians that infant's injuries were shaken baby syndrome, and not consistent with falling off a chair was sufficient for conviction of felony murder); State v. Leibhart, 662 N.W.2d 618 (Neb. 2003) (expert testimony on shaken baby syndrome admissible; passes Daubert); Order Denying Motion to Exclude Testimony on AHT/SBS at 5, State v. Mendoza, No. 071908696 (Utah Dist. Ct., June 5, 2009) ("[T]he State's experts made a very compelling... showing that SBS is both still widely accepted and applicable to the current case"); see also R v. Harris, [2005] EWCA (Crim) 1980, [267] (Eng.); R v. Henderson; R v. Butler; R v. Oyediran, [2010] EWCA (Crim) 1269, [7] (Eng.).

514 While other criteria, such as academic appointment, research, and publication, are desirable, they are not necessary to declare one as a part of the "relevant" scientific community.

515 See Kassirer & Cecil, supra note 54, at 1383 (discussing Kumho).

516 R v. Henderson; R v. Butler; R v. Oyediran, [2010] EWCA (Crim) 1269, [208] (Eng.) (emphasis added).

517 See, e.g., Martinez, 74 P.3d at 323; McClary, 541 A.2d at 102; State v. Edmunds, 746 N.W.2d 590, 593 (Wis. Ct. App. 2008); Order Determining Admissibility of Expert Testimony on AHT/SBS at 22-23, Commonwealth v. Davis, No. 04-CR-205 (Ky. Cir. Ct., Apr. 17, 2006); Order Denying Motion to Exclude Testimony on AHT/SBS at 6, State v. Mendoza, No. 071908696 (Utah Dist. Ct., June 5, 2009).

518 Compare Order Denying Motion to Exclude Testimony on AHT/SBS at 5-6, State v. Mendoza, No. 071908696 (Utah Dist. Ct., June 5, 2009) (accepting AHT testimony), with Edmunds, 746 N.W.2d at 594 (giving a new trial because scientific doubt surrounds AHT diagnoses).

519 See supra Section II.B.c.1--"A Shifted Consensus?"--where fifteen national and international medical societies are listed as publicly supporting the validity of AHT as a medical diagnosis. As mentioned in that section, the only "relevant" disciplines with some discord are pathologists and biomechanical engineers.

520 Daubert v. Merrell Dow Pharm., Inc., 509 U.S. 579, 590 (1993).

521 Kumho Tire Co. v. Carmichael, 526 U.S. 137, 152 (1999).

522 Breyer, supra note 57, at 6.

523 See Jerome P. Kassirer & Richard I. Kopelman, Learning Clinical Reasoning 16 (1991) ("Bayesian analysis assembles a complete set of diagnostic hypotheses that can explain a given set for clinical findings. For each hypothesis, a set of relevant attributes is identified (historical findings, physical findings, complications, predisposing factors, laboratory results) that might help discriminate among the diagnoses. The prior probability of each diagnostic hypothesis is specified numerically, as is the probability that each attribute is found in each disease entity. Then, a calculation is make of the likelihood of each disease entity given the disease prevalence and the

probability of each clinical attribute."). Although physician reasoning does not exclusively proceed in a Bayesian fashion, physicians do frequently rely on Bayesian reasoning (combining disease prevalence with their knowledge of frequency of signs and symptoms in a given disease) in the diagnostic process. See Henifin, et al., supra note 91, at 467.

524 See Jerome P. Kassirer & Frank A. Sonnenberg, The Scientific Basis of Diagnosis, in Textbook of Internal Medicine 14, 14-15 (William N. Kelley ed., J.B. Libbincott Co. 1989); Kassirer & Kopelman, supra note 523, at 3.

525 See Kassirer & Sonnenberg, supra note 524, at 14; see also Kassirer & Kopelman, supra note 523, at 16 (defining differential diagnosis).

526 See Kassirer & Sonnenberg, supra note 524, at 15; see also Kassirer & Kopelman, supra note 523, at 11 ("Hypothesis refinement is an evolving, sequential process of data gathering and interpretation."). Rather than exclusively relying on statistical data on disease prevalence to generate diagnostic hypotheses, the physician also utilizes "heuristics" (or shortcuts/rules of thumb) to make the task of information gathering manageable and efficient. Kassirer & Kopelman, supra note 523, at 4.

527 See Kassirer & Sonnenberg, supra note 524, at 15; see also Kassirer & Kopelman, supra note 523, at 11. ("Hypothesis refinement is an evolving sequential process of data gathering and interpretation."). Probabilistic reasoning is Bayesian-type reasoning where prior probabilities of diseases are considered and combined with a physician's knowledge of the frequency of signs and symptoms in a given disease and the probabilities of specific test information. These assist the physician in a probabilistic assessment of the most likely hypothesis. Causal reasoning "is a function of the anatomical, physiological and biochemical mechanisms that operate normally in the human body and the pathophysiologic behavior of these mechanisms in disease." See Kassirer & Kopelman, supra note 523, at 28. Physicians "are accustomed to use any reliable data to assess causality, no matter what their source.... Temporal proximity can be a potent factor in causal decision making...." Kassirer & Cecil, supra note 54, at 1384.

528 "Adequacy occurs when a "diagnostic hypothesis... encompasses all surviving hypotheses and... accounts for all the patient's findings, whether abnormal or normal." Kassirer & Kopelman, supra note 523, at 32. Coherency occurs "when a patient's findings are consistent with the altered pathophysiology of the hypothesized disease state." Id. Parsimony is "the simplest possible explanation all of the [patient's] findings." Id.

529 See Best v. Lowe's Home Ctrs. Inc., 563 F.3d 171, 179, 183-84 (6th Cir. 2009) (stating a differential diagnosis can be adequate grounds for a causation opinion under Daubert); Hyman & Armstrong, P.S.C. v. Gunderson, 279 S.W.3d 93, 107, 109 (Ky. 2008); Westberry v. Gislaved Gummi AB, 178 F.3d 257, 263 (4th Cir. 1999). But see, Moore v. Ashland Chem. Inc., 151 F.3d 269, 279 (5th Cir. 1998) (denying admissibility of expert testimony based upon the differential diagnosis); Moore 151 F.3d at 288 (dissent).

530 See Gunderson, 279 S.W.3d at 107 (citing Globetti v. Sandoz Pharms. Corp., 111 F.Supp.2d 1174 (N.D. Ala. 2000)).

531 In re Paoli R.R. Yard PCB Litig., 35 F.3d 717, 759 (3d Cir. 1994) (noting there is "a requirement that experts at least consider alternative causes" and that this concept is "at the core of differential diagnosis."); see Heller v. Shaw Industries, Inc., 167 F.3d 146, 156 (3d Cir. 1999) (stating that before allowing differential diagnosis reasoning as grounds for causation, a medical expert must rule out "obvious alternative causes," but not, "categorically, all other possible causes" of an injury).

532 See State v. McClary, 541 A.2d 96, 102 (1988) (noting shaken baby syndrome is generally accepted by medical science).

533 See United States v. Vallo, 238 F.3d 1242, 1245 (10th Cir. 2001); People v. Dunaway, 88 P.3d 619, 633-34 (Colo. 2004); People v. Martinez, 74 P.3d 316, 323, 324-25 (Colo. 2003); State v. Leibhart, 662 N.W.2d 618, 627-28 (Neb. 2003); State v. Glenn, 900 So.2d 26, 34-35 (La. Ct. App. 2005); Order Denying Motion to Exclude Testimony on AHT/SBS at 5-6, State v. Mendoza, No. 071908696 (Utah Dist. Ct., June 5, 2009).

534 R v. Harris, [2005] EWCA (Crim) 1980, [4]-[5].

535 Id. at [3].

536 Id. at [56].

537 Id. at [5].

538 Id. at [57]-[58] (emphasis added).

539 Id. at [68]-[69] (emphasis added).

540 See id. at [102]-[103].

541 Id. at [101].

542 Id. at [153].

543 Id. at [153], [266].

544 Id. at [185], [219].

545 Other challenges to admissibility of AHT testimony have included 403 challenges (that a medical diagnosis of child abuse is confusing to a jury in relation to the legal definition of child abuse, within a particular state, and consequently, the prejudicial value outweighs the probative value) and challenges to the admissibility of testimony on the amount of force required to cause the injuries. See People v. Martinez, 74 P.3d 316, 321-22 (Colo. 2003). For a comprehensive review of the evidentiary challenges in AHT testimony, see John E.B. Myers, Myers on Evidence in Child, Domestic, and Elder Abuse Cases (Aspen Publishers, vol. 1 2005) and John E. B. Myers, Myers on Evidence in Child, Domestic and Elder Abuse Cases (Aspen Publishers, supp. 2007).

546 See supra Section II(B)(2)("Statistical Evidence").

547 Mack et al., supra note 396, at 208.

548 Squier & Mack, supra note 396, at 10.

549 See id.; Mack et al., supra at 396, at 208.

550 See supra Part (d)(ii) Alternative Hypotheses.

551 A Local Auth. v. S, [2009] EWHC (Fam) 2115 [63], [199], [201]-[203] (Eng.) (emphasis added).

552 Id. at [284]-[286] (emphasis added) (heading omitted).

553 R v. Henderson; R v. Butler; R v. Oyediran, [2010] EWCA (Crim) 1269 [188], [190] (Eng.) (emphasis added).

554 State v. Smallwood, 955 P.2d 1209, 1220-21 (Kan. 1998).

555 See State v. Smith, 877 So. 2d 1123, 1127-29 (La. Ct. App. 2004) (fatal shaking and impact case; doctor testified child's injuries were abusive); State v. Smallwood, 955 P.2d 1209, 1221 (1998) (infant died of inflicted head injury; pathologist opined the child died of abuse: "by stating that, based upon her medical experience, Kaine died as a result of child abuse, either shaking or a blow to the skull, Dr. Gould was not testifying as to the ultimate question of Smallwood's guilt or innocence. Expert testimony in the form of an opinion is not objectionable because it embraces the ultimate issue or issues to be decided by the trier of fact.").

556 Estelle v. McGuire, 502 U.S. 62, 68 (1991).

557 Id. (citation omitted).

558 See Kempe et al., supra note 148, at 143; Tuerkheimer, supra note 4, at 31.

559 See State v. Torres, 121 P.3d 429, 446-47 (Kan. 2005).

560 State v. Edmunds, 746 N.W.2d 590, 596 (Wis. Ct. App. 2008).

561 See R v. Henderson; R v. Butler; R v. Oyediran, EWCA (Crim) 1269 at [188]-[190]; Order Denying Motion to Exclude Testimony on AHT/SBS at 1-4, State v. Mendoza, No. 071908696 (Utah Dist. Ct., June 5, 2009).

562 Order Determining Admissibility of Expert Testimony on AHT/SBS at 22, Commonwealth v. Davis, No. 04-CR-205 (Ky. Cir. Ct., Apr. 17, 2006).

563 See, e.g., State v. Leibhart 662 N.W.2d 618, 627-28 (Neb. 2003); Order Denying Motion to Exclude Testimony on AHT/SBS at 5-6, State v. Mendoza, No. 071908696 (Utah Dist. Ct., June 5, 2009).

564 See Edmunds, 746 N.W.2d at 596.

565 See id.

566 A Local Auth. v. S, [2009] EWHC (Fam) 2115 [199] (Eng.) (emphasis added).

567 Breyer, supra note 57, at 4.

568 Id. at 6.

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Articles

A DAUBERT ANALYSIS OF ABUSIVE HEAD TRAUMA/SHAKEN BABY SYNDROME--PART II: AN EXAMINATION OF THE DIFFERENTIAL DIAGNOSIS

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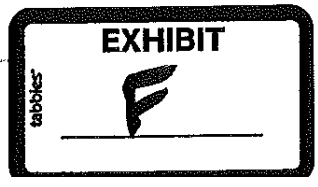

***205 I. Introduction**

For reasons inexplicable to many physicians, and unbeknownst to many others, the diagnosis of Abusive Head Trauma/Shaken Baby Syndrome [1] (AHT/SBS) remains a lightning rod for controversy. Public media articles continue to be published. [2] Legal articles ***206** continue to be written. [3] And judicial commentary on the science continues to occur. [4] The most recent example of judicial commentary upon the topic, and probably the most prominent, is the dissenting opinion of the honorable Justices Ginsburg, Sotomayor, and Breyer in Cavazos v. Smith. [5] In that opinion, the dissenting justices cited seven medical articles that ostensibly supported their opinions that: 1) "there was inadequate scientific evidence to come to a firm conclusion on most aspects of causation, diagnosis, treatment, or any other matters pertaining to SBS"; 2) "that 'the commonly held opinion that the finding of [subdural hemorrhage] and [retinal hemorrhage] in an infant was strong evidence of SBS was unsustainable'"; and 3) that "doubt has increased in the medical community 'over whether infants can be fatally injured through shaking alone.'" [6]

Setting aside the multiple concerns regarding the selection criteria for the articles, [7] the irony in the citation of these articles is that the articles cited by the dissenting justices are actually so methodologically flawed, scientifically inaccurate, and of the lowest level of evidence-based medical literature, that they would be reasonable examples of articles that are "not even good enough to be wrong." [8] So how do the most learned jurists in the land get the ***207** science so wrong? [9] And what hope is there then for the lone "gatekeeper"?

In Part I of this discussion, one of the authors, Dr. Narang, presented a relatively comprehensive analysis of the current science surrounding AHT/SBS, and more specifically, surrounding two of the most common injuries found in AHT/SBS--subdural hemorrhages (SDHs) and retinal hemorrhages (RHs). [10] Dr. Narang asserted that the diagnosis of AHT is supported by "at least 700 peer-reviewed, clinical medical articles comprising thousands of pages of medical literature, published by over 1,000 different medical authors, from at least twenty-eight different countries." [11] He described, in painful detail, multiple scientific studies from various medical disciplines that demonstrated a significant statistical association of SDHs with AHT [12] (over accidents and other medical causes) and that demonstrated a highly significant statistical association of severe RHs with AHT [13] (over accidents and other medical causes). Despite the reported "controversy" on the topic, Dr. Narang cited at least fifteen international and national professional medical societies that have publicly acknowledged the validity of AHT either through formal practice statements or through educational materials provided to their members or the public. [14] In conclusion, Dr. Narang examined that scientific literature with Daubert scrutiny and argued that such literature was scientifically valid, and

consequently, did provide the clinician with sound scientific basis for arriving at diagnosis of AHT. [15] However, as Dr. Narang alluded to in his initial article, that was only part of the analysis.

***208** Part II of this discussion swings the microscope in the opposite direction. The differential diagnosis of AHT (and its two most common injuries, SDHs and RHs) includes many things--accidental trauma, birth trauma, bleeding disorders, malignancy, and metabolic/genetic syndromes, to name a few. [16] Although a detailed scientific analysis [17] of the entire differential diagnosis is beyond the purpose and scope of this article, this paper will examine the best, current evidence-based data for two of the most common items on the differential diagnosis, accidental injury and bleeding disorders, and then examine the evidentiary basis for two of the most debated topics in AHT, biomechanics and hypoxia/ischemia. In being provided with the "rest of the story," the reader will, hopefully, be able to see the relative strengths and weaknesses of the scientific data underpinning SDHs, RHs, and the differential diagnosis. The reader will thereafter be able to discern for himself or herself whether the scientific data afford the clinician reasonable grounds for arriving at the diagnosis of AHT. More importantly, the reader will be provided with a reasonable glimpse of the entire analysis --the methodology--a clinician undertakes in arriving at the AHT diagnosis and be able to conclude for himself or herself whether that methodology is reasonable or simply "junk science."

In the first subsection of this paper, we briefly review the concept of "evidence-based medicine," discuss its proper role in present-day clinical medicine, and proffer an acceptable ranking scale for evidence-based medical literature, [18] a scale that shall be applied to the scientific literature discussed herein. Thereafter, we place accidental injuries and bleeding disorders under the evidence-based microscope, examining that literature in light of the Oxford rating ***209** scale for evidence-based medicine. We then detail the current state of knowledge on biomechanics and hypoxiaischemia, [19] highlighting the weaknesses and limitations that infect that literature. Finally, we magnify the microscopic examination of the differential diagnosis method, examining it closely with both a scientific and legal lens. In so doing, we garner and dissect the scientific and legal arguments around that methodology, discuss the fallacious arguments critiquing the AHT/SBS literature, and propose some solutions going forward for the identification and promulgation of sound scientific evidence on the topic in the legal setting.

II. Evidence-Based Medicine & AHT/SBS

"I look upon it as being a great part of the art to be able to judge properly of that which has been written." [20]

-Hippocrates

Physicians have been trained in the natural sciences, the advancement of medical knowledge, and the critical appraisal of medical literature since the dawn of medicine. [21] Medical journals and the peer review system now date back nearly 200 years. [22] With the burgeoning of medical publications and the advent of electronic indexing of the medical literature in the 1970s and 1980s, it clearly became untenable for an individual practitioner to remain aware of all the research activity in even a small specialty of medical practice. ***210** From this environment of rapid scientific discovery grew a new movement: Evidence-Based Medicine (EBM). Perhaps described best by one of its founders, Dr. David Sackett, EBM is the "conscientious, explicit, and judicious use of the current, best evidence in making decisions about individual care." [23] The review article containing an expert's opinion about research was replaced by a systematic review: a thorough, exacting, and repeatable methodology for grading and summarizing the most current medical evidence.

A central tenet of EBM is that not all evidence is of equal quality. [24] In seeking out the "best" evidence, one has to make value judgments, and do so without reference to the outcome of the study. [25] The value judgments that ascribe one study as better than another are primarily based upon the technical elements of the design and execution of the study. [26] There has been a proliferation of "rating scales" that rate medical studies. [27] Some scales [28] are more appropriate for assessing the quality of

literature on therapeutic modalities *211 in clinical medicine; others provide multiple scales that are amenable to different kinds of studies.[29] For example, the Centre for Evidence Based Medicine (CEBM) at The University of Oxford utilizes five different scales--for assessments of literature on therapy, prognosis, diagnosis, symptom prevalence, and economic decision analysis.[30] The CEBM scale for diagnostic literature is listed below in Table 1.[31]

Table 1: Centre for Evidence Based Medicine Levels of Evidence Scale for a Diagnosis[32]

1a	Systematic review (with homogeneity) of level 1 diagnostic studies or clinical decision rule with 1b studies from different clinical centers.
1b	Validating cohort study with good reference standards or clinical decision rule tested within one clinical center.
1c	A diagnostic finding so strong that it absolutely confirms or refutes the diagnosis.
2a	Systematic review (with homogeneity) of 2b or better studies.
2b	Exploratory cohort study with good reference standards; clinical decision rule after derivation or validated only on split-samples or databases.
3a	Systematic review (with homogeneity) of 3b or better studies.
3b	Non-consecutive study or without consistently applied reference standards.
4	Case-control study, poor, or non-independent reference standard.
5	Expert opinion without explicit, critical appraisal; or based on physiology, bench research, or "first principles."

*212 A prime example of the misapplication of ratings scales is that of Donohoe in his oft-cited, unfortunate article Evidence-Based Medicine and Shaken Baby Syndrome.[33] While Donohoe decries the absence of randomized-controlled trials,[34] the CEBM levels of evidence scale for a diagnosis does not even include randomized-controlled trials.[35] The CEBM correctly recognizes that the randomized trial, while excellent for evaluating a therapy, is not an appropriate tool for evaluating a diagnosis.

The last element of the "current, best evidence" in EBM is the emphasis on the adjective "current."[36] Some critics of the AHT/SBS diagnosis propose a level of diagnostic abstinence until some future level of scientific precision (akin to a "DNA-type" evidence) can be achieved.[37] Yet, even the most ardent EBM advocates would not purport such diagnostic impotence whilst awaiting some yet-unrealized, "absolute" diagnostic certainty. The emphasis of EBM is to focus on the best available evidence, not to discard the evidence we have simply because better evidence is not yet available.

*213 III. Accidental Injury

Accidents,[38] often as a result of falls, are a commonly reported cause of head injury in children.[39] In Fujiwara et al.'s review of 28 AHT cases and 232 non-abusive head injuries, fall was the history presented in at least 17.9% and 62.9% of the cases, respectively.[40] This section details the evidence that physicians can reliably utilize to distinguish accidental from non-accidental trauma. While it might seem that accidental trauma and inflicted trauma would be difficult to differentiate, a number of studies demonstrate that this actually is possible.

A. Short Falls

A particularly common childhood injury reported to pediatricians is the "short fall."[41] Because injuries later thought to be abusive are often blamed on short falls, they merit special attention. Authors in pediatric literature have defined short falls as heights varying from less than 15 feet,[42] to 10 feet,[43] to less than 1.5 meters *214 (4.9 feet).[44] Although there exists no standardized definition of a "short fall," more recently consensus has shifted toward recognizing a "short fall" as a fall of less than 1.5 m.[45]

There is an abundance of medical literature on pediatric short falls. Studies have focused on a variety of injury aspects--whether there are observed differences in witnessed versus unwitnessed falls, in falls of varying degrees of height, and in falls with varying biomechanical forces and aspects.[46] Study designs have ranged from isolated case reports to large epidemiologic studies and systematic reviews.[47] Methodological differences have posed challenges to collectively assimilating data in systematic reviews.[48] However, a comprehensive review[49] of the scientific literature does permit the clinician to draw the following conclusions with a reasonable degree of medical certainty.

Scientific Conclusion #1: When caregivers have been surveyed, they report that short falls are common, but severe[50] injuries from *215 short falls are rare.

In 2010, Suzanne B. Haney et al. surveyed 307 parents, asking if their child had fallen off a "high surface" such as a table, bed, or dresser, before the age of two.[51] Forty percent of parents recalled such a fall, and fifty-nine percent of parents recalled more than one such fall.[52] Among the 209 reported falls, the only serious injuries reported were two concussions.[53] There were no reported subdural hematomas, retinal hemorrhages, or deaths.[54] These results accorded with Warrington et al., a similar, but much larger, study from the United Kingdon in 2001.[55] In that study, when their child had reached six months of age, parents were asked to describe any accident that had occurred with their child since birth.[56] The authors received data on over 2500 children, with over 3300 falls being reported.[57] Of these approximate 3300 falls, 1782 (53%) were falls from beds or settees.[58] Only twenty-one falls (less than one percent) resulted in concussion or fracture.[59] There were no reported intracranial injuries *216 or death.[60] Other authors who have surveyed parents/caregivers have obtained similar results.[61] Although these two cross-sectional studies would merit only a 4 on the Oxford CEBM scale,[62] they hold some scientific significance in that the subjects studied had no motivation or inclination to provide inaccurate data.[63]

Scientific Conclusion #2: Short falls occurring in objective settings, such as hospitals, have not resulted in subdural hematoma or death.

In 1993, Lyons and Oates reported on 207 children who fell out of bed in the hospital in which a nurse either observed the fall or attended to the child within seconds of the fall.[64] Falls ranged from 32-54 inches.[65] One child sustained a skull fracture and another child sustained a clavicle fracture.[66] There were no multiple injuries, visceral injuries, severe head injuries, or deaths.[67] These results accorded not only with prior studies by Nimityongskul[68] in 1987 and Levene[69] in 1991, but with subsequent studies by Monson[70] in 2008, *217 Ruddick[71] in 2010, and Schaffer[72] in 2012. In total, these case series describe over 620 falls in objective[73] settings (i.e., hospitals), with no consequent serious head injuries or deaths. Although these studies are case series, or level 3b evidence on the CEBM scale, given the increased level of objectivity to the observed injuries, they warrant significant scientific consideration.

Scientific Conclusion #3: Children in large, licensed daycares rarely die from short falls.

Another source of independently observed childhood falls is daycare. Licensed daycare centers are an attractive environment to study short falls because structural layout and institutional policy effectively minimize abuse and because of facilitates' reporting of accidents and abuse.[74]

Chadwick conducted a comprehensive review of all daycare studies in the world's medical literature.[75] He found twenty-five studies that focused on injuries occurring in daycare, studies from the U.S., Canada, Sweden, Norway, and Denmark.[76]

Only two of the twenty-five studies specifically looked at deaths in the daycare setting.[77] Good et al. reviewed data on over 520,000 children in day- *218 care centers and found no deaths attributed to falls.[78] Wrigley and Dreby examined data on over six million children in child care centers over an eighteen-year period (1985-2003) and found only two reported deaths attributed to falls in large, licensed child care centers.[79] However, these two reported deaths stemmed from newspaper reports and, consequently, provided no detailed data for scientific analysis.[80] Despite having over 900,000 children under age two in U.S. daycare centers[81] with multiple falls occurring daily, to date there is no peer-reviewed medical report of a death resulting from a short fall in a large, licensed daycare center.[82]

Scientific Conclusion #4: Severe injuries and deaths are rare in short falls witnessed by two or more adults, but ironically, are more common in short falls witnessed by a single adult.

Williams reviewed the cases of 398 patients treated with injuries resulting from a fall.[83] Of these 398 patients, 106 were less than three years old and the fall was witnessed by at least two adults.[84] The only death in this group was a child who fell seventy feet.[85] The only serious injuries resulting from observed falls less than ten feet were children (three percent) with depressed skull fractures after falling against an "edged" surface.[86] In contrast, among fifty-three children *219 under three whose falls were witnessed by fewer than two adults, eighteen had serious injuries and two died after reported falls of less than five feet.[87] Similar results and conclusions were reached by Wrigley and Dreby[88] in their comprehensive review of daycare literature, by Reece & Sege,[89] and by Johnson et al.[90] Although Williams's, Johnson's, and Reece & Sege's case series only merit level 3b evidence on the CEBM scale, they provide interesting insight into the histories provided by caregivers in abusive and accidental injuries.

Scientific Conclusion #5: If reports of deaths from uncorroborated short falls are accepted as valid, then short falls appear to be more dangerous than longer falls.

Williams noted another anomaly. In his study of 398 patients, uncorroborated reports indicated two deaths followed falls of less than five feet, but no deaths followed falls of 6-11 feet or 12-23 feet.[91] Chadwick et al. noted similar findings in their review of 317 uncorroborated falls requiring medical attention.[92] Among 183 children who reportedly fell 5-45 feet, only one died.[93] Among 100 children reported to have fallen less than four feet, seven died.[94] All seven children had other factors concerning for a false history, including old fractures, bruising on the trunk or extremities, genital injury, or *220 more than one impact point on the head.[95] Similar results were obtained by Reece & Sege.[96] Although the biomechanics of a short fall are a complex phenomenon, and probably not simply a factor of height,[97] other authors have also noted a correlation between increased fall height and injury severity.[98]

Scientific Conclusion #6: Well-designed prospective studies reveal that severe injuries or deaths resulting from short falls are rare events.

In 1992, Duhaime et al. prospectively studied 100 patients less than two years of age who suffered head injuries.[99] In efforts to avoid "circularity" concerns, Duhaime et al. used strict criteria for determining "inflicted" injury.[100] The authors excluded retinal hemorrhages (RHs) as a diagnostic criterion and only included SDHs that had no history of trauma but had clinical or radiologic findings of blunt impact to the head.[101] Thus, the authors designed an algorithm that was "deliberately biased to reduce false positives and, thus, underestimate the true incidence of child abuse."[102] In the Duhaime et al. cohort, seventy-six patients' injuries were determined to be from *221 accidental causes, and twenty-four were determined to be inflicted.[103]

Of the 100 studied patients, seventy-three suffered head injuries from reported falls, nine from motor vehicle accidents, two from impacts by other objects, two admitted assaults, and fourteen without any history. [104] Of those seventy-three reported falls, thirty-four (47%) were from falls less than four feet, twenty-one (29%) from falls greater than four feet, and eighteen (25%) from falls from walkers or down stairs. [105] Of the thirty-four reported short falls, twenty-six were determined to be accidental, and eight were determined to be inflicted. [106] Of the twenty-six accidental short falls, none had SDHs or RHs. [107] In Duhaime et al.'s entire 100-patient cohort, there were only four deaths--three from the inflicted group and one from the accidental group. [108] The only death from the accidental category was a passenger in a high-speed motor vehicle accident. [109] Similar results were obtained by Bechtel et al. in their prospective study of 87 children aged 0-2 years at Yale Children's Hospital from 2000-2002. [110]

In 2011, Thompson et al. reported their prospective study of seventy-nine children less than four years of age who presented to the emergency department of Kosair Childrens' Hospital (Louisville, KY) between May 2008-July 2009 with a complaint of a household fall from a bed, sofa, or similar furniture. [111] The authors sought to determine the severity of injuries that resulted from accidental short-distance household falls in children and to investigate the association *222 of fall environment and biomechanical measures with injury outcomes. [112] The authors excluded all children suspected of abuse and included only children whose injuries were "definite" or "likely" accidents. [113] The authors conducted interviews with the caregivers and in-depth scene investigations in all seventy-nine cases in order to obtain information regarding fall dynamics and to determine biomechanical measures associated with these falls. [114]

Of the seventy-nine subjects enrolled, fifteen had no injuries, forty-five had minor (AIS 1) injuries, [115] seventeen had moderate (AIS 2) injuries, [116] and two had serious (AIS 3) injuries. [117] No subjects had injuries classified as AIS 4 or higher, and there were no fatalities. [118] The authors also determined that, in their study, "furniture height, impact velocity, and child BMI were found to have the greatest influence on injury severity outcomes. Children with moderate or serious injuries tended to have fallen from greater heights, had greater impact velocities, and had a lower BMI than those with minor or no injuries." [119] Thompson et al. concluded that "[t]his study provides a comprehensive evaluation of the biomechanics of short-distance household falls and investigates the association of biomechanical and fall environment measures with injury severity. Children aged 0-4 years involved in a short-distance household fall did not sustain severe or life-threatening injuries." [120] Duhaime's, Bechtel's, and Thompson's studies merit level 2b evidence on the CEBM scale.

***223 Scientific Conclusion #7: Systematic reviews of the short-fall literature indicate that short falls rarely cause death in children.**

In 2009, Chadwick et al. sought to numerically quantify the risk of death from short falls. [121] The authors performed an extensive review of the published medical literature on short falls, "including 5 book chapters, 2 medical society statements, 7 major literature reviews, 3 public injury databases, and 177 peer-reviewed, published articles indexed in the National Library of Medicine." [122] The authors examined data and literature from any and every short fall aspect: reliably witnessed falls, child-care studies, studies of large clinical populations (more than fifty cases), studies of single cases or small series (less than fifty cases), studies of long falls, pathologic and cadaveric studies, studies involving biomechanical analysis, playground-fall studies, studies involving falls down stairs, walker-related falls, parent-observation studies, and studies specifically addressing short-fall death. [123]

When reviewing the two large injury databases (California's EPIC [124] database and the CDC's WISQRS [125] database), Chadwick et al. found that WISQRS allowed determination of the total fall death rate for children less than four years of age, but

did not provide stratification according to fall type/height. [126] Per the WISQRS database, "[t]he 'all fall' death rate was [three] cases per [one] million young children per year." [127] However, California's EPIC database did stratify data for short falls. [128] For the period 1999-2003, the EPIC database revealed six short fall fatalities. [129] In a state with 2.5 million ***224** children under five years old, this calculated to 0.48 deaths per million children per year. [130] Chadwick et al. provided comparative risk estimates for other conditions that caused death in infants and children (see Figure 1 below):

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Figure 1: Table from David L. Chadwick et al., Annual Risk of Death Resulting [131]

Hence, based upon Chadwick et al.'s data, the chance of any given child dying of a short fall in any given year is approximately one in two million. However, to truly approximate the probability of a single fall causing death, one would need to multiply the one in two million by the number of falls a typical toddler or child experiences in a year.

Ehsani et al. also conducted a comprehensive review of the short-fall literature with the aim of answering the question: "Can a simple short fall cause fatal head injury in an infant?" [132] Toward this aim, ***225** Ehsani et al. considered 1055 publications for inclusion. [133] Using explicit selection criteria, only twenty-seven publications were included in their review. [134] The authors concluded that it is "rare, but possible, for fatal head injury to occur from a simple short fall." [135] The authors went on to state that "[l]arge population studies of childhood injuries indicate that severe head injury from a short fall is extremely rare. This is counter pointed by a single documented case report that demonstrates it can happen." [136]

Plunkett also sought to assess the plausibility of short-fall deaths. [137] He reviewed the National Electronic Injury Surveillance System (NEISS) and determined that, over a twelve-year period (1988-1999), eighteen children had fallen from playground equipment and subsequently died. [138] Plunkett's data, however, suffer from several limitations. First, Plunkett's study is not a true systematic review of the short-fall literature. Second, the NEISS database Plunkett reviewed suffers from selectivity bias. [139] Finally, Plunkett's reported deaths are inaccurate. [140]

The Chadwick et al. systematic review of the short-fall literature constitutes level 2a evidence on the CEBM scale, whereas Plunkett's ***226** study constitutes level 3b or 4 evidence.

B. Other Accident Literature

In addition to the short-fall literature, several well-designed prospective studies comparing accidents and abuse cases have identified clinical variables that can discriminate accidents from abuse case with a high degree of statistical significance. The strength of these studies lies in their ability to validate prior exploratory studies with good reference standards, thus constituting level 1b evidence on the CEBM scale. For example, Vinchon et al.'s 2010 prospective series of eighty-four patients who sustained injuries from either witnessed accidents (N=39) or confessed inflicted head injury (N=45-- obtained from judicial sources) determined the specificity and positive predictive value of severe RHs for abusive injury to be 97% and 96%, respectively. [141] This validated Vinchon et al.'s and Bechtel et al.'s prior exploratory prospective studies in 2005 and 2004, respectively, where the authors found high statistical significance and specificity of more severe RHs for abuse. [142]

In enhancing prior research efforts, Hymel et al. developed a national, multi-site research collaborative, Pediatric Brain Injury Research Network (PediBIRN), that is dedicated to conducting rigorous clinical research on pediatric traumatic brain injury. [143] The strengths of the studies produced by this collaboration include the prospective multicenter design, the breadth and depth of data capture, and the a priori application of criteria for abusive and non-abusive causes (criteria that are specifically designed to

minimize circular reasoning and inherent biases). [144] In 2010, the PediBIRN *227 group published the results of its prospective multicenter study that examined the diagnostic, prognostic, and forensic significance of depth of intracranial injury in accidental and non-accidental cases. [145] After thoroughly reviewing data on fifty-four children less than three years old at nine sites, the authors found that children "with sub-cortical injuries (i.e., injuries deeper in the brain) more frequently had been abused" (odds ratio [OR]: 35.6; P-value <0.001) than had suffered accidents. [146] Although this particular study constitutes level 2b evidence on the CEBM scale, its rigorous methodology has provided the foundation for the development of emerging higher levels of evidence--clinical prediction rules. [147]

Finally, in 2009, Maguire et al. completed a systematic review of the world's medical literature and identified clinical features that differentiate accidental from non-accidental head injury in child-ren. [148] The authors conducted "[an] all-language literature search of [twenty] electronic databases, websites, references, and bibliographies from 1970-2008." [149] Using over 100 keyword combinations, this yielded 320 studies for review. [150] Applying strict inclusion and exclusion criteria, [151] the authors determined that fourteen studies were appropriate for inclusion, which represented 1655 children: 779 with inflicted brain injury (iBI) and 876 with non-inflicted brain injury (niBI). [152] The authors utilized multi-level logistic regression analysis *228 to arrive at positive predictive values and odds ratios for various clinical features. [153]

As a result, the authors found that "apnoea appears to be a critical distinguishing feature (PPV for abuse 93%, OR 17.06)." [154] This means that, in the comparative diagnostic subset of accidental versus non-accidental injury, a child who arrives at the hospital not breathing is seventeen times more likely to have been abused. Likewise, the authors found retinal hemorrhages "were strongly associated" with inflicted brain injury, with a PPV of 71% and an OR of 3.5. [155] The authors stated, "[a] child with an intracranial injury who has co-existent retinal haemorrhages [sic] is significantly more likely to have iBI than niBI." [156] The authors concluded, "[t]his review is the largest of its kind, and offers for the first time a valid statistical probability of iBI when certain key features are present (e.g., retinal haemorrhage)." [157] Maguire et al.'s systematic review comprises level 2a evidence on the CEBM scale.

C. Conclusion

The scientific literature on pediatric accidental injury is robust, sound, and constantly improving. No longer is the isolated case report [158] (level 4 evidence) or the poorly designed case series [159] (level 4 *229 evidence) a sufficient basis for scientific conclusions. Currently, there is level 1b and 2a evidence supporting clinicians in the distinction of abusive from accidental injury when certain clinical features, such as apnea or extensive/severe retinal hemorrhages, are present. And physicians continue to build upon these data, with hopes of soon attaining the highest level of evidence attainable--level 1a. In a recent analysis, Maguire et al. estimated the probability of AHT based on six clinical features, reporting that combinations of the above factors are even more predictive. [160] For example, the authors determined that a child (less than three years old) with a subdural hematoma plus any three of the following factors: apnea; retinal hemorrhage; rib, skull, or long-bone fractures; seizures; or head or neck bruising; had a positive predictive value for AHT of greater than 85% and an odds ratio of greater than 100. [161] Armed with such data and analysis, physicians today can confidently conclude that certain infants' injuries are the result of intentional injury and are not the result of accident. And they do so on the basis of the highest quality medical evidence.

IV. Bleeding Disorders

Bleeding disorders may be proposed as the underlying cause for clinical findings in cases of suspected abusive head trauma (AHT). Unlike some hypothesized "mimics" of AHT, [162] bleeding disorders are known causes of intracranial hemorrhage (ICH) and retinal hemorrhage and are a heterogeneous group of conditions that vary in etiology, presenting symptoms, and

prevalence. [163] All three of these *230 descriptive characteristics must be considered when evaluating the potential for a bleeding disorder to be the cause of an ICH.

It is important to remember that: 1) only certain bleeding disorders may cause findings that may be confused with AHT; 2) most bleeding disorders are rare; 3) the more common bleeding disorders typically are mild; and 4) ICH resulting from bleeding disorders is a rare complication of the more severe diseases. [164] The probability of a rare disorder causing an even rarer manifestation (i.e., ICH) is the main scientific consideration when there is concern for a potential bleeding disorder in a young child with an ICH and a history of no or minimal trauma. [165]

A. Causes (Etiologies) of Bleeding Disorders

Bleeding disorders may be congenital (inherent to the genetic makeup of an individual) or acquired. Congenital bleeding disorders may cause symptoms from the time prior to birth to anytime throughout a person's lifetime. [166] Bleeding symptoms may occur at any time and, depending on the severity of the bleeding disorder, may be asymptomatic (without symptoms) for long periods of time. [167] Acquired bleeding disorders are the result of a condition that is not permanently engendered in a person. [168] As with congenital bleeding disorders, symptoms may present at variable times during one's life, and their severity and duration are dependent upon the specific condition. [169]

*231 1. Congenital Bleeding Disorders

Examples of congenital bleeding disorders include hemophilia and von Willebrand disease (VWD). There are many other congenital bleeding disorders, [170] and significant variability exists in the prevalence and presenting symptoms of each of the bleeding disorders, such that each disorder must be considered individually. A discussion of the specific congenital bleeding disorders is outside of the purpose and scope of this review. [171]

2. Acquired Bleeding Disorders

Acquired bleeding disorders may occur at any age, may be isolated, or may occur due to medications, medical illnesses/ conditions, or trauma. [172] Specific examples include immune throm bocytopenic purpura (ITP), [173] disseminated intravascular coagulation (DIC), [174] vitamin K deficiency bleeding (VKDB), [175] and liver coagulopathy. [176] Often these acquired bleeding disorders are transient, but *232 some may persist. They either affect platelet number or function or result in a coagulation factor deficiency.

In large part, a clinician can identify acquired causes of bleeding by taking a careful history, performing a detailed physical examination, and ordering the appropriate laboratory tests. For example, a variety of medications can lead to platelet dysfunction (e.g., non-steroidal anti-inflammatory drugs, sodium valproate). [177] A careful medication history can evaluate for this potential. [178] Other acquired bleeding disorders, such as ITP or other causes of thrombocytopenia (low platelet count), can be readily diagnosed on the basis of a lab test--a complete blood count (which manifests the low platelet count). [179] DIC is evident on other laboratory testing--a prolonged prothrombin time (PT), a prolonged partial thromboplastin time (PTT), decreased fibrinogen level, and elevated D-Dimer levels. [180] In VKDB, laboratory tests show a prolonged PT and a normal PTT. [181] When testing for the specific coagulation factors that are dependent on Vitamin K (factors II, VII, IX, and X), they are markedly decreased. [182] If findings and initial laboratory testing is concerning for VKDB, but vitamin K treatment has already been provided, measurement of proteins induced by vitamin K absence can confirm the diagnosis. [183]

The prevalence of ICH in people with acquired bleeding disorders is variable but very low. For example, the prevalence of ICH in patients *233 with idiopathic ITP is <1%. [184] In VKDB, administration of oral vitamin K prophylaxis reduces the incidence of late VKDB from 4.4-10.5/100,000 live births to 1.5-6.4/100,000 live births, and all patients with VKDB do not have ICH. [185]

B. Symptoms of Bleeding Disorders

Although bleeding disorders, by definition, cause bleeding, the manifestations of bleeding disorders vary based upon location on the body, frequency, and severity. Some bleeding disorders, such as the mild platelet abnormalities and VWD, generally cause mild symptoms, such as mouth and/or nose bleeding or mild skin bruising. [186] Often, mild platelet abnormalities and VWD cause no symptoms at all. [187] More severe conditions, such as some types of hemophilia, often cause severe joint bleeding and may cause ICH. [188]

When young children present with ICH and no history of trauma or a history of a minor trauma, one must consider a bleeding disorder as the underlying cause. ICH occurs more frequently in some bleeding disorders, very rarely in others, and either exceedingly rarely or not at all in other bleeding disorders. [189] As will be discussed below, the prevalence of each bleeding disorder and the prevalence of ICH within the population of people with that specific bleeding disorder may be used to identify a testing scheme to evaluate for bleeding disorders as a cause of ICH. However, prior to using the existing *234 data to construct an evidence-based approach to evaluating for bleeding disorders in the setting of alleged AHT, it is necessary to first examine the data for validity and applicability.

C. Sources of Data

Nearly all of the existing data regarding bleeding disorders and ICH have been culled from large databases, such as the Universal Data Collection (UDC) database project of the Centers for Disease Control. [190] The UDC was established in 1997 to monitor the safety of the blood supply in the United States and to track the incidence and consequences of joint complications in patients with bleeding disorders. [191] The UDC and other similar databases were not constructed to guide forensic evaluations regarding possible AHT and bleeding disorders. As a result, the details of the bleeding symptoms in the databases have not been collected in a fashion that would be preferable for forensic purposes. [192] For example, specifics such as trauma history, location of ICH (subarachnoid/subdural, etc.), and external evidence of trauma have not been collected. However, this does not eliminate the utility of the UDC and other databases in forensic consideration.

The existing scientific literature generated from these hematology databases is useful in determining the prevalence of particular bleeding disorders in our population and the probability of a particular bleeding disorder to cause ICH in general. Even in the absence of large studies evaluating the forensic implications of *235 bleeding disorders, the existing literature of case series and case reports is valuable. [193] In the largest study examining non-accidental injury and bleeding disorders, Jackson et al. documented presentations of bleeding disorders over a ten-year period at a large pediatric center. [194] After excluding patients diagnosed at birth with a bleeding disorder, 15.3% of all children with bleeding disorders presented in a manner that may be confused with abuse, including genital and buttock bruising, bruising in immobile infants, and ICH. [195] Five children in the study were involved with the legal or child protection system due to bleeding/bruising. [196] The authors concluded that bleeding disorders can present in a manner that is "clinically indistinguishable from abuse." [197]

However, of note, only two children over the study's ten-year interval presented with ICH, both of whom were older than one year of age, had obvious evidence of impact (skull fractures), and VWD. [198] The authors were unable to determine if the

findings in these cases were due to abuse or accidental impact. [199] There were no cases of spontaneous ICH (with no evidence of impact) in the study. [200] This suggests that spontaneous ICH due to a bleeding disorder is an extremely rare event.

***236 D. Testing for and Probability of Bleeding Disorders in the Setting of ICH**

Studies have evaluated the use of screening questions and family history in the detection of bleeding disorders. [201] Negative screens (i.e., the family or child has no history of easy bleeding/bruising) are not effective ways of ruling out a bleeding disorder. [202] Similarly, statements such as "my child bruises easily" or a family history of "easy bruising" do not rule out abuse as a cause of bleeding in a child. [203] Thus, when clinicians consider the potential need for testing for bleeding disorders, in the absence of a known, named bleeding disorder in the child or family, the family history of bleeding/ bruising is of limited use.

When ordering laboratory tests for clinical or forensic reasons, clinicians must ask, "What is the potential for a positive test result?" and "How is the result of this test going to change my forensic impression or patient management?" If the potential for a positive test result is microscopically small or if a positive test result does not change the clinical impression/diagnosis, there is very little value in sending the test. For instance, the prevalence of Factor 2 (pro-thrombin) deficiency (one per one million people) and frequency of ICH within the population of people with Factor 2 (prothrombin) deficiency (11%) make testing for Factor 2 (prothrombin) deficiency of extremely low value on cases of suspected AHT. [204]

Because there is a remote potential for a bleeding disorder presenting as ICH, clinicians often consider evaluating for such in cases of possible AHT. [205] However, when clinically assessing a particular child, the entire set of clinical and historical findings must be ***237** considered together. [206] If a child has other findings that are highly suggestive of violent trauma (e.g., fractures) or other findings that are unrelated to bleeding disorders, it is reasonable to exclude testing for bleeding disorders. [207] An evaluation for bleeding disorders is generally performed if a child has ICH with no readily apparent explanation or with no other evidence strongly suggesting abuse (e.g., unexplained fractures, witnessed abuse, patterned bruising) as this may rarely be the presenting manifestation of a bleeding disorder. [208]

A basic tenet of practicing evidenced-based medicine is that actual evidence rather than hypotheses can be used to guide clinical decision making. [209] Hypothetical considerations without proven cause-effect linkage to ICH, such as vaccines [210] and "choking episodes," [211] cannot be considered evidence-based medicine. Thus, testing for histamine levels or vitamin C levels based on a hypothesis that vaccines induce ICH by altering levels of those factors is not grounded in scientific, evidence-based practice. [212] Such hypotheses should be tested by rigorous prospective research prior to being offered as an explanation for ICH in a legal setting.

When deciding on which tests to order to evaluate for a bleeding disorder as the cause of ICH in a young child, clinicians can access the existing data on the prevalence of specific bleeding disorders and the prevalence of ICH due to those specific bleeding disorders. If the prevalence of a condition and the frequency of a particular presentation of that condition are known, a physician can construct the probability of that specific condition (bleeding disorder) resulting in the specific presentation (ICH):

***238** $PTP(B) = P(A) \times P(B|A)$ [213]

For example, severe VWD is extremely rare, occurring at an upper-limit estimated population prevalence of 1 per 300,000 people. [214] Up to four percent of people with severe VWD initially present with a "head bleed," including both ICH and extracranial (scalp or facial) bleeding. [215] Thus, the estimated probability that a person will get an ICH due to severe VWD is: (Prevalence of severe VWD) x (Prevalence of ICH in severe VWD)

(1/300,000) x (0.04) = 1/7.5 million

The calculated probability, in this instance, is actually an overestimate of severe VWD causing subdural hemorrhage (SDH) because the calculated probability pertains to not only all ICH (SDH, subarachnoid hemorrhages, and epidural hematomas and parenchymal bleeding[216]), but scalp and facial bleeding as well. Additionally, spontaneous SDH (i.e., those not resulting from trauma) is a subset of all SDHs. Thus, it is reasonable for a clinician to conclude that the chances a young child will suffer SDH (either spontaneously or as a result of trauma) from a previously undiagnosed, severe VWD are exceedingly small.

Similar probabilities can be calculated for any bleeding disorder in which: 1) the prevalence of the condition is known and 2) the ***239** prevalence of ICH within the condition is known (See Table 1, infra).

Table 2: Probabilities for Congenital Coagulopathies to Cause ICH[a,b] [217]

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***240** [a] The probability of having a specific bleeding disorder increases in the setting of a family history of that specific-named bleeding disorder or if the patient is from an ethnicity in which a specific bleeding disorder is more common (e.g., Ashkenazi Jewish people and factor XI deficiency).

[b] "Probability" indicates the probability that an individual in the general population would have the following specific coagulopathy causing an intracranial hemorrhage.

For instance, the two most common severe congenital bleeding disorders, Factor 8 and Factor 9 deficiencies (two forms of hemo-philia), have probabilities for ICH of 1 per 50,000 males and 1 per 200,000 males, respectively.[218] The probability of Factor 13 deficiency causing an ICH is 1 per 6 million, largely due to the rarity of Factor 13 deficiency.[219] This means that, in the population in general, a single person's risk of having an ICH due to Factor 13 deficiency is 1 in 6 million.

Ordering tests to evaluate for every bleeding disorder is generally impractical due to the statistical implausibility of many of the bleeding disorders causing ICH. In fact, many conditions have a probability of causing ICH so low as to preclude calculation. However, these probabilities can be used to identify the need for tests to be ordered if a child's findings may reasonably be caused by a bleeding disorder. Thus, physicians may order tests with higher (relatively speaking) probabilities, such as evaluating for conditions that have a probability higher than or equal to 1 in 5 million, for instance. If negative test results are obtained, the post-test probability of one of the tested bleeding disorders is essentially zero.

E. Special Considerations

Two bleeding disorders have particular potential to create confusion or pose a diagnostic challenge in the evaluation of possible ***241** AHT--von Willebrand disease and mild platelet disorders.

1. Von Willebrand Disease (VWD)

VWD is the most common congenital bleeding disorder.[220] It most commonly presents with mild to moderate bleeding from the nose or mouth, bruising, or heavy bleeding during a woman's menstrual period.[221] It is generally classified in terms of von Willebrand factor (VWF) levels and the type of functional defect affecting the VWF protein.[222] Type 1 disease results from an absolute decrease in the VWF protein and is the most common.[223] Type 3 is characterized by nearly absent levels of VWF as well as low factor 8 and is the most severe version of VWD.[224] There are also a number of qualitative abnormalities

resulting in variable bleeding manifest-ations (types 2A, 2B, 2M, and 2N).[225] Testing for VWD can be complicated and often requires consultation with a pediatric hematologist.[226]

The current prevalence of VWD is difficult to determine,[227] as recent consensus changes have resulted in more specific diagnostic criteria.[228] Per the National Heart, Lung, and Blood Institute, the ***242** most recent criteria for diagnosis requires VWF levels <30% (normal range 50-200%), resulting in fewer individuals with levels below the normal range meeting diagnostic criteria.[229] Current estimates indicate that low VWF levels may occur in up to 1% of the U.S. population.[230] However, since many persons with VWD do not manifest symptoms, the prevalence of symptomatic persons with VWD is currently best estimated at 23-113 per million or 0.0023-0.01% of the U.S. population.[231]

Additionally, and more importantly, ICH as the presenting finding of severe VWD is extremely rare (upper limit of probability of 1 per 7.5 million people).[232] Mild VWD is much more common than severe VWD, but the prevalence of ICH within people with mild VWD is unknown.[233] Because low VWF levels are relatively common, it is certain that testing for VWD will be positive in a small percentage of children with findings concerning for AHT.

In nearly all cases, where VWD is mild, it often does not cause any symptoms until a hemostatic challenge, like the removal of teeth.[234] A review of the peer-reviewed literature in humans on ICH due to VWD reveals four published cases of spontaneous ICH in adults.[235] None of these cases involved subdural hemorrhage.[236] The peer-reviewed medical literature evaluating mild trauma and VWD in humans contains only a few published case reports and case series of VWD complicating mild or more severe trauma in mobile children or ***243** adults.[237] Only one of these cases involved subdural hemorrhage--that case being a four-year-old child who suffered an impact to the head.[238] There are currently no scientific data to support the hypothesis that VWD is a cause of spontaneous ICH in young, immobile children. And the best current scientific literature supports the conclusion that VWD may very rarely contribute to bleeding complications in mild head trauma.

The clinician is posed with a diagnostic challenge when the historical and clinical findings are consistent with AHT, but laboratory testing shows low VWF levels. Some individuals have concluded that laboratory tests consistent with VWD essentially eliminate the consideration of AHT.[239] Others have been more balanced, stating that "[t]he significance of von Willebrand disease as a possible contributory factor in infants with subdural and retinal hemorrhages should be further addressed."[240] The notion that laboratory testing consistent with VWD "rules out" AHT as a diagnosis is not only irrational (as the presence of VWD does not protect a child from AHT), but is also unsupported by the scientific literature.[241]

***244** 2. Mild Platelet Disorders

Congenital platelet disorders can result in fewer platelets, abnormal function of platelets, or a combination of the two.[242] Mild congenital platelet disorders include Quebec platelet disorder, the MYH9 related disorders, Scott syndrome, Hermansky-Pudlak syndrome, Chediak-Higashi syndrome, and Wiskott-Aldrich syndrome.[243] Specific testing for platelet function is required to detect these disorders.[244] Most bleeding with these disorders is mild and manifests as excessive bruising or menorrhagia (heavy menstrual periods).[245]

The exact prevalence of mild platelet disorders is unknown.[246] The probability of mild platelet disorders causing ICH is also unknown but is likely very low given the typical clinical manifestations.[247] Much like VWD, if specific testing is performed in children with suspected abusive ICH, it is likely that a small number of children will have laboratory results indicative of a mild platelet disorder.[248] In these cases, laboratory results do not rule out AHT, as there are currently no scientific data to support the hypothesis that mild platelet disorders have caused a spontaneous ICH.[249]

Rare severe congenital platelet disorders, such as Bernard-Soulier *245 syndrome (BSS) and Glanzmann thrombasthenia (GT) are known causes of ICH.[250] In both of these disorders, significant mucocutaneous bleeding and ICH have been reported, although ICH is rare, occurring in only 0.3-2% of patients with GT and even less in those with BSS.[251] Screening for these disorders may be accomplished using the Platelet Function Analyzer test (PFA-100).[252]

F. Conclusion

The evaluation of level of evidence for the probability that a bleeding disorder caused an ICH is a "symptom prevalence" question. The most recent Oxford Centre for Evidence-Based Medicine levels of evidence ratings[253] do not address these types of questions. However, the 2009 version of the Oxford Centre for Evidence-Based Medicine does address "symptom prevalence" questions and is an appropriate evaluation tool.[254] There are a large number of studies evaluating the potential for ICH in patients with specific bleeding disorders with variable levels of evidence.

Any studies based on prospective registries, such as the Universal Data Collection of the Centers for Disease Control and Prevention and the North American Rare Bleeding Disorder Registry, qualify as "1b" according to the 2009 Oxford levels.[255] Most other studies on the subject are best classified as 1c and 2b. Any individual claiming that an ICH might be the result of a "bleeding disorder" should specify which bleeding dis-order is of concern, the general prevalence of that bleeding disorder *246 in the population, and the probability that that specific bleeding disorder causes ICH.

V. Biomechanics

Classical mechanics began with the work of Isaac Newton in the late 1600s.[256] Beginning with a few simple equations, engineers can predict how many objects will respond to various forces.[257] One branch of mechanics, biomechanics, concerns itself with "the scientific study of mechanics in biological systems."[258]

In contrast to the observational, clinical studies noted in the accidents section above, biomechanics is fundamentally an experimental discipline. Biomechanical engineers, like all responsible scientists, are unwilling to injure living children in the course of an experiment. Instead, engineers employ a number of approximations, ranging from animals to constructed "crash test dummies" to finite element analysis (FEA). Each of these techniques has some value, and none is perfect. The best scientific insight results from a careful consideration of the strengths and limitations of all available information.

Traumatic brain injury can result from either inertial (a rapid head acceleration-deceleration that produces injurious brain deformation) or contact (where impact produces local brain deformations) mechanisms.[259] Although rapid brain acceleration can proceed in either linear or rotational directions, it is actually high angular accelerations and velocity that are often correlated with intracranial hemorrhage and severe brain injury.[260]

*247 Traditionally, biomechanical analysis of head injury in infants and children assumed that infants and young children responded like small adults.[261] Using an engineering approach called "dimensional analysis," it was assumed that critical inertial loading conditions for severe brain injuries (such as SDHs or diffuse axonal injury) could be scaled from adults to infants based solely upon brain mass.[262] However, recent biomechanical studies[263] have shown what pediatricians have long argued --that children are not small adults. Multiple differences--in tissue composition, brain and skull properties, and brain vulnerability--between adults and children have prompted scientists to interpret biomechanical studies that utilize scaling approaches with caution.[264]

The biomechanics of AHT/SBS is a comprehensive and complex topic. A few biomechanical questions are frequently encountered in AHT/SBS. We will summarize the scientific literature pertinent to those questions. For a more comprehensive discussion of the topic, we refer the reader to additional literature. [265]

Question 1: Does the biomechanical literature suggest that shaking alone cannot cause SDH or serious brain injury in children?

As Narang mentioned in his first article, when traumatic, SDHs are caused by rupture of the bridging veins in the brain. [266] Early biomechanical work by Gennarelli and Thibault in primates revealed a strong association of SDH with elongation of the bridging veins beyond their strain tolerances when the primate brains moved relative to their skulls during sudden acceleration-deceleration ***248** events. [267] In 1987, Duhaime et al. published a biomechanical study of shaken baby syndrome in which the authors shook a constructed model infant. [268] Duhaime suggested that impact was necessary to cause SDH because shaking alone achieved maximum velocities and accelerations that were well below the thresholds for SDH (that were scaled from adult primates) and impact exceeded those thresholds. [269] This study created a tide of misplaced sentiment that shaking alone could not cause significant injury in infants and children. [270]

Scientific critique of the Duhaime study has highlighted the importance of biofidelity in doll models. Cory and Jones [271] found that making just minimal adjustments to Duhaime's model (such as altering the center of gravity in the head) created a model in which manual shaking did exceed injury thresholds in eight out of ten trials. [272] Wolfson et al.'s FEM study determined that slight modifications of the stiffness and hinge used in the model's neck dramatically altered the rotational accelerations and velocities achieved. [273] These studies underscore the principle that even the slightest variation in the experimental model can result in aberrant data.

Further studies have shown that other factors not considered in Duhaime's study also affect the likelihood of injury. Eucker showed that the direction of head rotation (back and forth versus side to ***249** side.) affects likelihood of injury. [274] Prins et al. suggested that both adult and infant brains are increasingly susceptible to repetitive injury (i.e., implying a cumulative effect of injury). [275] Finally, Kochanek and colleagues have mounted evidence that biochemical and metabolic responses to brain injury are significantly different in the young infant compared to the older child or adult. [276]

In 2003, Prange et al. performed an updated version of Duhaime's study. [277] Like Duhaime's prior study, Prange et al. compared biomechanical forces achieved from shaking and shaking with impact (which ended with forceful impact onto a rigid or padded surface but without throwing the model). [278] The authors concluded that "[v]igorous shakes of this infant model produced rotational responses similar to those resulting from minor falls, but inflicted impacts produced responses that were significantly higher than even a 1.5-meter fall onto concrete." [279]

Contrasting Duhaime's and Prange's findings, a host of biomechanical studies have yielded different conclusions. In an FEM study by Roth et al., the authors questioned whether angular acceleration--the value for which Duhaime and Prange compared injury thresholds--could accurately predict injury. [280] Roth et al. re-created the injury events in the Prange study using a computational ***250** model. [281] In spite of dramatic differences in rotational acceleration, shaking and impact created similar strain on the bridging veins. [282] Because shaking and impact cause similar strains on the bridging veins, it is not unreasonable to expect similar injuries to result. [283]

A valuable FEM study by Morison examined the protective effect of cerebrospinal fluid (CSF) on intracranial injury. [284] The CSF is an important but difficult to model structure that is notably absent from Duhaime's and Prange's models. [285] Morison

demonstrated that the CSF dampens brain acceleration to 0.13-1.00% of translational skull acceleration but does not protect the brain from rotational acceleration.[286] Duhaime's and Prange's studies did not incorporate or study Morison's assertion that cerebrospinal fluid protects the brain against a short fall but not from shaking.

Finally, Finnie et al. studied shaking in actual living animals.[287] The authors grasped 7-10-day-old lambs under the axilla and vigorously shook them.[288] The lamb model was selected principally because it has a relatively large gyrencephalic brain, large head, and weak neck muscles resembling a human infant.[289] No lamb suffered ***251** impact to the head.[290] Each lamb was shaken ten times for thirty seconds.[291] This methodology was based upon perpetrator confessions that indicate that repeated and violent shaking is common in AHT.[292] Two of the seven lambs shaken in this manner suffered small SDHs,[293] and two lambs had minor retinal hemorrhages.[294] Shaken lambs showed significantly more damage on microscopic examination compared to lambs that were not shaken.[295]

To honestly answer the question whether shaking alone can cause SDH and/or severe brain injury, the answer must assess all of the studies mentioned above, not just the Duhaime study of 1987. As discussed above, numerous authors have identified variables not considered by Duhaime that are important determinants of injury. Perhaps the most pointed criticism of these anthropometric model studies for determining injuries from shaking comes from the Prange paper itself:

> These injury projections should be interpreted with caution, because differences in species, age, material properties, geometry, and direction make scaling experimental angular acceleration and velocity measurements to infants problematic when based on differences in brain mass alone. To avoid the limitations of using scaled loads from animal and cadaver experiments to investigate real life events, case studies of minor falls in infants were also used to examine injuries that occur as a result of falling from different heights. Unfortunately, these falls are rarely witnessed, load measurements of the event are lacking, contact surface information is rarely given, and the population studied generally includes a broad age range, rather than just newborns.[296]

In conclusion, in pediatric head injury, it must be remembered ***252** that there are no human data on load tolerances[297] causing SDHs. What has been presented above is that the biomechanical literature does not offer a definitive "yes" or "no" answer to the widely debated question of whether shaking alone can cause SDHs. Some literature demonstrates it can occur, while other literature disputes it. It is clear, however, that continued assertion of the principle--that biomechanics clearly demonstrates that SDHs and/or serious brain injury cannot result from shaking--is disingenuous and scientifically irresponsible.

Question 2: Does biomechanics show that injurious shaking would necessarily cause catastrophic neck injury?

A 2005 paper by Bandak is widely cited as additional biomechanical evidence supporting the proposition that shaking alone cannot cause the injuries noted in AHT.[298] In that paper, Bandak proposed that shaking sufficient to cause brain injury would necessitate devastating injuries to the cervical spine.[299] Bandak's paper is purely analytic; meaning, rather than conducting original experiments, the purpose of the study was to reinterpret previously published data.[300] Reinterpretation of published data is a valid study design and is the design of several of the studies discussed previously.

Bandak relied upon Jenny's[301] reports of rotational acceleration and Duhaime's[302] report of rotational velocities from adults shaking a model infant.[303] Using these data, Bandak computed the amount of ***253** force experienced by the neck during these shaking events.[304] Bandak's computed forces greatly exceeded prior estimates of neck tensile strength in several animals.[305]

Because children with AHT are frequently not noted to have severe neck injuries, Bandak concluded that a reevaluation of AHT is needed.[306]

This work has suffered much scientific criticism. Three of the four studies of neck tensile strength[307] do not involve humans and were presented with an explicit condition that they were preliminary and not to be used as references.[308] The remaining article[309] studied static rather than dynamic loads imposed by a shaking baby.[310] Other authors noted that neck injury is actually not as rare in AHT as Bandak asserted.[311]

The most serious criticism, however, is that Bandak's computations are simply incorrect. Stated simply, Bandak's math is wrong. This is especially notable because a purely analytic study should be perfectly replicable.[312] When nine other scientists (including two well-reputed biomechanical engineers whom Bandak himself cited in his original paper) attempted to repeat Bandak's mathematics, they found numerous ***254** gross errors.[313] When these scientists repeated the computations themselves, they found that the correct value for every single neck force was at least ten times lower than the values reported.[314] They determined that the corrected values do not exceed the threshold for neck injury.[315] Confirming Bandak's errors, a second group of scientists independently attempted to replicate Bandak's work and produced results identical to the first.[316]

In his response, Bandak admitted that the reported values did not result from the equations published in the paper.[317] Bandak suggested instead that he "basically integrated the [AHT] accelerations over the time duration of shaking,"[318] without providing any of the equations, data, or assumptions necessary to replicate the work.[319] So, when asked to produce a single "worked example" demonstrating how the reported forces could be computed, Bandak failed to do so.

Replication is a fundamental mechanism by which scientific validity is achieved. A work that cannot be replicated isn't bad science--it isn't science at all. Bandak's suggestion that shaking sufficient to cause injury would necessarily cause neck injury is a prime example of how invalid biomechanical data have been misused in court.[320] Further reliance upon these data or citation to this work should be avoided.

Question 3: Has biomechanics shown that skull fractures are likely *255 to result from a short fall?

As discussed in the Accident section above, short-distance falls are a common presenting history in children thought to be physically abused.[321] Several studies have demonstrated that the fetal and infant cranial bone increases in stiffness with age.[322] Coats and Margulies studied the material properties of infant cranial bone and suture in order to better predict the outcome of infant falls.[323] They obtained donated skull and suture materials from twenty-three fetuses and infants ranging from twenty-one weeks of gestation to thirteen months of age.[324] The authors measured the elastic modulus[325] in infants and found that the adult cranial bone modulus was thirty times higher (less deformable) than a one-month-old infant's.[326] A one year old's cranial bone modulus was eighteen times higher (less deformable) than a one-month-old infant's.[327] Additionally, the pediatric suture[328] deforms 30 times more than pediatric skull bone and 243 times more than adult bone before ***256** rupture.[329] These large strains in the pediatric skull and suture may result in large-scale deformation upon impact and explain the diminished frequency of intracranial injury with short falls.

Weber, a German pathologist, conducted two studies on infant cadavers in order to see how frequently fractures would occur on a variety of surfaces at changing table height.[330] In the first study, the cadavers of fifteen infants, no older than 8.2 months of age, were dropped (five each) from eighty-two centimeters (thirty-two inches) onto a stone-tile floor, a carpeted floor, and linoleum backed with foam flooring.[331] All of the infants had pathologic conditions that did not involve the head, and no skull

fractures existed before testing, neither on palpation nor on skull radiographs.[332] All the subjects made simultaneous contact with their back and parieto(side)-occipital(back) region of the head.[333] All fifteen of the cadavers sustained fractures and, in three cases, linear fractures crossed suture lines.[334]

In his second paper, Weber tested an additional thirty-five cadavers with age ranges from newborn to 9.1 months at time of death.[335] Ten were dropped in the same manner and at the same height (thirty-two inches) onto a foam mat two centimeters thick and twenty-five were dropped onto a doubly folded blanket eight centimeters thick.[336] One of the ten (10%) infants dropped onto the rubber mat sustained parietal (side of the head) fractures that did not cross ***257** sutures.[337] However, four out of twenty-five (16%) infants dropped onto the blanket sustained parietal skull fractures.[338]

However, in contrast to Weber's data, Snyder et al. found different results.[339] Using newspaper clippings to screen for cases of free-falls in a six-state area, investigators were sent out to verify that the falls were, in fact, free-falls unimpeded from reaching the landing site by intervening obstacles and that the landing site had not changed since the time of the fall (e.g., construction or destruction of a building, change in soil or sand consistency due to significant rains).[340] One-hundred-ten free-fall cases were thoroughly investigated (age, biometrics, height of fall, landing surface, initial landing posture, and medical outcome).[341] Twelve cases were subsequently simulated in detail, using a computer simulation (MVMA 2-D Crash Victim Simulator, Version 3) that had been validated.[342] Seven of the twelve simulations involved head-first impacts; among these were five children who were younger than four years old and who fell 10'6" -34'2" .[343] All of these children had skull fractures, concussion, or both.[344] Upon examination of injuries as a function of impact surface for head-first fall cases, only one child who fell from a height of approximately twelve feet did not have a skull fracture.[345] Snyder concluded that the tolerances for skull fracture in infants and toddlers without pathology were between 4-10 feet (i.e., after a four-foot fall some would have a fracture, and after a ten-foot fall virtually all would have a skull fracture).[346]

***258** Bertocci et al. used an anthropometric model to simulate a three-year-old child rolling off a twenty-seven-inch "bed" onto playground foam, carpet, linoleum, and wood.[347] Despite acknowledging "a paucity of injury criteria for children," Bertocci concluded that the risk of a contact head injury (essentially a skull fracture) resulting from a short rolling fall is "low."[348]

Coats and Margulies conducted a finite element model (FEM) study using parametric simulations of occipital impacts to predict the likelihood of occipital or parietal skull fractures.[349] The authors found that elements arrays one standard deviation above the mean ultimate stress of occipital or parietal bone gave an 84.1% chance of occipital or parietal fracture.[350] At three standard deviations above the mean ultimate stress of the parietal bone, there was a 99.8% likelihood that the parietal bone would fracture.[351] They found such a condition at the equivalent of an eighty-two centimeter fall onto concrete and concluded that they had good validation with Weber (1984, 1985).[352]

The accident literature quoted earlier also suggests that short falls can result in skull fractures but at rates far less than those ***259** reported by Weber.[353]

Thus, the biomechanical literature demonstrates that short falls can result in skull fractures. However, in spite of Weber's data, it would be unreasonable to suggest that all, or even a majority, of short falls cause skull fractures because multiple, well-designed clinical studies document that the vast majority of short falls do not result in a skull fracture.

Question 4: Has biomechanics shown that SDHs are likely to result from a short fall?

The Prange study mentioned above simulated a child being dropped onto concrete, carpet, and a foam mattress.[354] For the drop experiments, the model was suspended at 0.3 meters (one foot), 0.9 meters (three feet), and 1.5 meters (five feet) over the landing surface.[355] The model was suspended with the head slightly below the remainder of the body, such that the head would strike the ground first.[356] The investigators concluded that 0.3-meter falls were unlikely to cause subdural hematomas but were unable to comment on the likelihood of injury in the 0.9-meter and 1.5-meter falls due to uncertainty about injury tolerances.[357]

Thompson et al. simulated a twelve-month-old child falling feet first onto wood, carpet, playground foam, linoleum over wood, and linoleum over concrete.[358] They simulated falls from zero, nine, and twenty-nine inches, measured from the dummy's feet.[359] The authors concluded "the risk of severe head injury for a [twelve]-month-old ***260** child in feet-first free falls across all tested surfaces and heights was low."[360] A similar study by some of the same authors comparing falls onto wet linoleum to dry linoleum found similarly low risks for femur (large bone in the upper part of the leg) fracture or head injury.[361]

In another study, Thompson et al. visited the homes of children who presented to the hospital for treatment after an accidental short fall.[362] The investigators specifically excluded cases where there was concern for possible child abuse.[363] Similar to prior studies, the experimenters found serious injuries to be rare and found no critical or life-threatening injuries.[364] Additionally, the surface the child fell on, pre-fall position, post-fall position, and motion prior to the fall all failed to demonstrate a statistically significant effect on injury severity resulting from the fall.[365]

The short-fall reconstruction articles cited here represent a field in its infancy. They represent reconstructions of only a handful of seemingly limitless permutations of fall height, impact surface, initial position, fall biomechanics, and dummy characteristics that are involved in simulating a short fall. Ongoing and future research on the topic will most likely refine our current knowledge on the specific biomechanical parameters that will cause SDHs.

Question 5: Does the biomechanics literature show that shaking can cause retinal hemorrhages?

Rangarajan et al. constructed an FEM of an infant eye based on ***261** head CTs of six normal, young children.[366] The model showed that rhythmic shaking significantly increases the stress on the retina.[367] The strongest forces were found at the posterior pole and the periphery.[368] Notably, as Narang discussed in his first article, this is where retinal hemorrhages in AHT are often found.[369]

A similar model by Hans et al. demonstrated that force on the retina during a single shaking incident is about thirteen times that of a short fall with a head impact.[370] A four-cycle shaking event produces forces on the retina fifty times that of a short fall.[371] The forces on the retina exceeded the adhesive strength of adult monkey retinas for the shaking, but not the short fall simulations.[372] These results coincide with clinical studies suggesting that retinal hemorrhages are more common in inflicted than accidental trauma.

Despite these findings, some learned biomechanical engineers suggest caution in interpreting these FEM studies.[373] They note that material properties, tissue-tissue interactions, and injury tolerances have not been measured or published for the pediatric eye.[374] They comment that, as with all biomechanical studies, a finite element analysis is only as good as its inputs and "inaccuracy of these inputs will yield fallacious outputs."[375]

***262** Animal studies have wrought further data on this topic. Coats et al. subjected 3-5-day-old piglets to a single, abrupt head rotation using a mechanical apparatus.[376] The piglets were kept alive for six hours, euthanized, and then autopsied.[377] Out

of fifty-one animals, four (eight percent) had retinal hemorrhages.[378] Similarly, as mentioned above, Finnie et al. noted minor retinal hemorrhages in two of seven sheep who were manually shaken.[379]

In summary, as with biomechanical studies on short falls, the data are preliminary and limited but enlightening. The current state of knowledge is that biomechanical studies support clinical evidence that retinal hemorrhages can be caused by shaking.

Conclusion

Scientists have a peculiar fondness for data. Data are the recorded results of the most fundamental scientific skill: careful observation. Good science welcomes valid data of all kinds and from all disciplines--dropping test dummies in a lab, shaking immature animals, and counting injured children as they come through the ER. The wise scientist recognizes that all data are flawed in some way. However, data are not discarded upon the identification of a single flaw. They are assessed in totality while carefully balancing the limitations of those data and, consequently, according appropriate weight to the compilation of all data. The biomechanical literature discussed here is in its infancy and, in spite of its flaws, is useful and informative. For anyone to assert blanket superiority of one type of data over another type (such as clinical data) is not just scientifically irresponsible; it is scientifically arrogant.

*263 VI. Hypoxia

As noted earlier, one of the alternative explanations proposed for the findings seen in victims of AHT is "hypoxia." Broadly, the term "hypoxia" is used to refer to a low level of oxygen in the body.[380] As a plastic condition, hypoxia's impact on the body depends upon a number of factors,[381] but broadly, it is important to think of hypoxia as both magnitude and duration. A small amount of hypoxia (slightly lower level of oxygen in the body) for a long duration has a very different effect on the body than profound hypoxia for a short duration.[382] It is important to recognize that "hypoxia" is not a single clinical or physiological entity and to speak of it as such is at best imprecise, and at worst simply wrong. This must be borne in mind as we unpack the role of "hypoxia" as a potential explanation for the finding seen in AHT.

For the past decade, some authors have proposed that "thin film" SDH, retinal hemorrhages, and acute encephalopathy (brain injury) can all be explained as being caused by hypoxia alone absent trauma.[383] Evolving from the hypoxia theory have been two adjunct hypotheses: (1) "dysphagic" choking[384] and (2) cervical spine (neck) injury[385] as causative of hypoxia, and thus, of the findings seen in *264 AHT. We will now review the published reports and clinical data used to support these proposals, often referred to as the "Unified Theory." It is important to note that in much of the literature used to support the hypoxia hypothesis, hypoxia as a clinical entity is undefined, making true analysis and comparison imprecise at best and misleading at worst.

A. Geddes and the Unified Theory

One of the cornerstones of the Hypoxia Hypothesis is a series of three papers by the British neuropathologist Dr. Jennian Geddes.[386] These have been often characterized as "Geddes 1,"[387] "Geddes 2,"[388] and "Geddes 3."[389] Broadly, they are used to characterize the "Unified Theory" of hypoxia as the main (or sole) cause of the spectrum of findings associated with AHT. We will now describe them in some detail, as they are misunderstood by many and, thus, are often improperly cited.

Geddes 1:

In 2000, Geddes and colleagues published a descriptive report on a non-sequential series of infant and child fatalities.[390] Given the paucity of prior published literature on the topic, the authors sought to analyze and report upon the neuropathologic findings in fifty-three cases of infants and children who had suffered inflicted brain injury.[391] They identified this cohort of fatalities using very similar ***265** criteria,[392] which many other authors and investigators have in the past and still currently utilize.[393] The investigators were not blinded to the cause or manner of death, nor were they blinded to the neuropathologic findings.[394] All clinical and legal records were reviewed and all brains were similarly systematically sampled and stained.[395]

Of these fifty-three subjects, thirty-seven were infants (under one year of age).[396] Half (n=27, 51%) had significant extracranial injuries e.g., burns, bruising, and/or fractures.[397] The majority (n=45, 85%) had signs of impact, including nineteen (36%) with skull fractures.[398] The authors reported that forty-four (81%) had subdural hemorrhage (SDH) with thirty-four being "thin film" (which was not defined by the authors).[399] Of the thirty-eight subjects in which a pathologist examined the eyes, the authors report that twenty-seven (71%) had retinal hemorrhages.[400] When compared with those without SDH, the authors reported that the presence of RH was statistically significantly ***266** associated with the presence of SDH ($p < 0.001$).[401] The authors reported that of the ten subjects without SDH, five did not have RH.[402] However, they also reported that they examined the retinas in only half of the subjects without SDH.[403]

In eight subjects that the authors called "shaken-only" by virtue of the absence of findings of cranial impact, five (of the six with examined eyes) had RH and seven presented with collapse or respiratory arrest.[404] The authors reported the most common microscopic finding was "global neuronal hypoxiaischaemia," seen in 84% of the infants and 63% of the older children.[405] Only three (6%) of the subjects had diffuse axonal injury.[406] The authors found no differences between the pathologies of subjects with and without evidence of impact.[407] The authors reported three significant clinical differences between infants and older children in their cohort: 1) infants had more apnea,[408] 2) infants had fewer extracranial (outside the skull) injuries,[409] and 3) infants had less subscalpular bruising (evidence of head impact).[410]

Geddes 2:

In their second paper, the same authors selected the same thirty-seven infants from their previous cohort, to which they added fourteen ***267** "control" infants for comparison.[411] The control group was a non-sequential group of infants who apparently died from non-abusive causes.[412] The authors reported on the intracranial histology[413] of these subjects and compared them with the infants utilized in their earlier cohort.[414] Again, the authors were blinded neither to the clinical information nor to the ultimate histopathologic findings.[415]

The authors reported that, of the thirty-seven cases of abusive head trauma (AHT), twenty-five (68%) had evidence of #APP staining (a stain conventionally associated with traumatic injury)[416] in axons.[417] None of the control infants were reported to have #APP staining identified in their brains.[418] They also reported that twenty-nine (78%) of the cases of AHT had widespread hypoxic neuronal injury, while only one infant (7%) in the control group had histologic evidence of severe hypoxia.[419] As noted earlier, twenty-one infant cases of AHT (70% of those examined) had bilateral RH and nine cases did not have RH.[420] Seven cases were not examined.[421] The comparison group did not have its retinal findings described.[422]

***268** The authors concluded that their "findings strongly suggest that severe traumatic axonal damage is a rarity in infant NAI unless there is considerable impact and that the diffuse brain damage responsible for loss of consciousness in the majority of cases is hypoxic rather than traumatic."[423]

Geddes 3:

Dr. Geddes and a similar set of co-authors then focused their attention to the dural covering of the brain.[424] They assembled fifty non-sequential fetal, neonatal, and infants who had non-traumatic deaths.[425] How this cohort was selected was not described.[426] To this cohort of cases, the authors compared three selected cases of AHT.[427] The authors did not describe how they chose these three infants, only that they were part of their earlier cohort.[428] Once again, the authors were blinded neither to the clinical information nor to the histologic findings.[429]

Of the fifty subjects, forty-one (82%) were either fetal or neonatal deaths, with seventeen (34%) intrauterine deaths and sixteen (33%) perinatal (younger than seven days of life) deaths.[430] Of the fifty cases, twenty-six (52%) died from hypoxia, eight (16%) from infection (not defined) with severe hypoxia (not defined), and six (12%) from infection (not defined) without hypoxia.[431]

Evaluation of the dural covering revealed only one subject (2%) ***269** with a macroscopic (seen without a microscope) SDH.[432] This infant was a twenty-five-week gestation whose mother had chorioamnionitis (infected placenta) and died from sepsis (blood infection).[433] The authors reported that thirty-six (72%) of the remaining cases had intradural hemorrhage (IDH).[434] This is described as "bleeding inside the strips of dura."[435] Retinas were not examined.[436]

The authors hypothesized that the IDH noted in their sample was, in essence, a precursor of the larger SDH,[437] which would be typically associated with trauma.[438] Despite reporting that there was no statistically significant relationship between hypoxia and IDH,[439] the authors extrapolated that the microscopic IDH could be caused by hypoxia (alone or with other factors) and, thus, larger SDHs could be caused by hypoxia (with or without other factors) as well.[440] Furthermore, despite no physiological data, the authors theorized that "cerebral venous hypertension and congestion, arterial hypertension and brain swelling, coupled with immaturity and hypoxia related vascular fragility" contributed to a cascade of events leading to findings similar to those seen in AHT.[441] Additionally, without reporting ophthalmologic findings, they hypothesized that RHs occur from a similar mechanism.[442]

B. Scientific Critique of the Unified Theory

The series of papers by Geddes et al. attempted to address two ***270** main themes. Geddes 1 and Geddes 2 attempted to describe the histopathologic findings in infants and children who are victims of AHT. They asserted that the histopathologic findings associated with AHT are similar to that seen in hypoxia-associated deaths. Geddes 3 attempted to describe how SDH could occur in the absence of trauma, utilizing hypoxia as the primary culprit. Clearly, the use of fetal and perinatal deaths obscures any conclusions on IDH or SDH because these are common findings at baseline (as outlined below).

Given the unblinded nature of the Geddes 1 and Geddes 2 subjects, true interpretation of the implications of the results must be guarded. A recent study of higher and more rigorous methodology involved three parallel assessors who were blinded to the clinical information.[443] The subjects were 24 child fatalities from a variety of causes (including AHT).[444] The reviewers were asked to assess the histo-pathology (#-APP) and indicate any evidence of trauma.[445] These blinded and independent assessors rated five of the seven child homicides as "trauma" and fifteen of the seventeen controls as "non-trauma."[446] This indicates that while not perfect, the histopathologic findings are exceedingly informative in determining the presence or absence of trauma. The authors rightly indicated that #-APP is clearly associated with trauma but urged caution saying, "the utility of #-APP is

quite powerful if not confounded by global hypoxicischemic injury, [and] ultimately, #-APP studies should be only one piece of information in the determination of cause and manner of death."[447]

Another notable aspect of the Geddes 1 and 2 papers is how their subjects were identified. The authors used inclusion criteria which included confession, conviction (with or without additional injuries), and discrepancy between findings and history provided (without adjudication).[448] Criteria like these have been utilized for decades by *271 many other researchers.[449] It is scientifically and logically inconsistent for some authors to criticize these other papers as having flawed inclusion methodology and yet cite the Geddes papers as "having important clinical implications"[450] or "landmark."[451] If the conclusions of the Geddes papers are sound and meaningful, then so are the conclusions of other studies which utilize the same methodology. One cannot have it both ways.

With regard to Geddes 3, there are two major obstacles in interpreting this paper. First, the authors present clinical information and data with significant imprecision. For example, indicating that an infant died from an "infection" or simply had "hypoxia" without clinical specificity leaves these terms vague and uninterpretable. "Infection" could be meningitis,[452] sepsis,[453] pneumonia, or pyelonephritis[454]; all of which are distinctly different medical entities. "Hypoxia," as noted above, is a heterogeneous clinical designation *272 that could be profound or trivial. Without clinical parameters, one cannot interpret the implications or potential signs or symptoms of the hypoxia.

Second, none of the authors' findings are new. The presence of intradural hemorrhage[455] (IDH) associated with fetal demise has been known for decades, if not centuries.[456] The authors themselves, citing Chase,[457] note "early study documented intradural bleeding as a 'constant finding' in premature infants."[458] In fact, one of the seminal monographs on birth-related injuries by Schwartz[459] from 1961 reports this history in the study of intracranial findings in the neonate. In summarizing the understanding of findings seen in fetuses and neonates, Schwartz writes "hemorrhage affects the dural reduplications (falx and tenotium) rather frequently."[460] In this context, the Geddes et al. report of IDH in a cohort of mostly fetal and perinatal deaths is neither new nor illustrative.

The presence of macroscopic, radiographically apparent SDH due to birth is also a well-described finding. An atlas by Cruveilhier in 1831[461] contains drawings of extensive meningeal hemorrhage over the hemispheres and, according to Schwartz,[462] Cruveilhier reported that these hemorrhages occurred in "at least one-third of neonatal *273 deaths." This is similar to the frequency (~ 25%) with which SDH has been reported in the past decade on neuroimaging of healthy term neonates.[463] It is striking that Geddes et al. reported only one (2%) subject with gross SDH. This rate is statistically different from the one-third reported in the mid-1800s or the one-quarter reported in the 21st century (chi square, p=0.000 and p=0.001).[464] This calls into question whether the entire cohort used by Geddes et al. in Geddes 3 is systematically different and not representative at all of "typical cases," either living or dead.

Lastly, and most importantly, if this model of hypoxia as outlined by Geddes et al. is indeed a reasonable explanation for an SDH similar to the ones associated with AHT, why were they not seen in their own study? The only SDH reported was in a fetus that did not have hypoxia, but instead sepsis, reported as a cause of death. In their study, all infants with hypoxia identified as a factor in their death did not have an SDH. If this model had fidelity to a true underlying pathophysiologic process, it would be expected that the finding they are attempting to explain would be present in at least some of the cases; it was present in none.

To truly assess whether hypoxia alone can result in SDH, one must use subjects that are more reflective of victims of AHT.[465] Infant and child victims of drowning or near drowning represent a nearly ideal population. The insult is pure hypoxia, the ages are *274 similar, the event is (sadly) common, the timing of the event is usually clear and not under dispute, and the outcome can be either fatal or non-fatal.

Rafaat and colleagues reviewed all infants and children admitted to the Children's Hospital of San Diego over a seventeen-year period who were on a drowning registry.[466] Of the 961 infants or children identified, 156 had a head CT scan within twenty-four hours of admission (some having two scans). Sixty-one were under three years of age, which was similar to other epidemiologic studies on drowning injuries. Fifty-eight scans had an abnormality identified. None of the abnormal scans had any SDH noted.[467] Thus, out of 156 children with a spectrum of hypoxia insults and clinical outcomes (including forty-one deaths), none had an SDH on the head CT scan. This would mean that, at most, hypoxia causes SDH two percent of the time, but it may be as rare as never.[468] This result accorded with Taylor et al.'s radiologic study in 1985[469] and Byard's[470] and Hurley's[471] pathology studies in 2007 and 2010, respectively.

Additionally, multiple lines of research have demonstrated that hypoxia is not a putative factor in causing RHs. Pitetti et al. prospectively studied 128 children less than two years of age who presented with apparent life threatening events (ALTEs) to determine the presence of *275 RHs.[472] Seventy-three of the 128 (57%) children received dilated fundoscopic exams. Only one child (1.4%) had RHs.[473] Upon further investigation, that case was determined to be a case of confessed abuse. Odom et al. prospectively examined the prevalence and character of RHs in patients in a pediatric ICU who had received at least one minute of chest compressions and survived.[474] After using strict exclusion criteria (such as excluding patients that had evidence of trauma, documented retinal hemorrhages before CPR, or suspicion of child abuse), Odom et al. found forty-three patients who met criteria for their study. Of the forty-three patients, "[t]he mean duration of chest compressions was 16.4 minutes . . . with 58% lasting between [one] and [ten] minutes."[475] All patients survived, and the authors found small punctate retinal hemorrhages in only one patient (2.3%). No patient had severe RHs. Finally, numerous animal studies investigating the effect of hypoxia on the retina have failed to demonstrate RHs.[476]

C. Adjunct Hypotheses

There are at least two parallel hypotheses to hypoxia that have recently been proposed. These hypotheses are "dysphagia/choking" or "coughing" and "neck injury." The "dysphagia/choking" or "coughing" hypothesis proposes that when an infant or a child coughs or gags, there is an increase in cerebral (brain) vascular pressure. This increased pressure, in the presence of hypoxia, causes rupture of cerebral blood vessels.

*276 Initially proposed by Talbert[477] in a non-peer-reviewed journal, Medical Hypotheses, it has since been promulgated by a few other authors.[478] Talbert has proposed that SDHs result from hypoxia and choking/coughing related to pertussis,[479] pyloric stenosis (stomach obstruction),[480] and/or gastroesophageal reflux with choking.[481] Talbert and Geddes subsequently attempted to replicate these physiologic conditions utilizing a mathematical software model of an infant.[482] Talbert and Geddes asserted that their model "has supported clinical observations, showing that the conditions necessary for subdural and retinal bleeding do occur in paroxysmal coughing, although it cannot prove that the bleeding is necessarily or even actually present clinically."[483]

The only other literature support for this hypothesis stems from a 2010 case report by Barnes and colleagues.[484] In that case, the authors report on a 4.5-month-old infant who was reportedly found by his father choking and blue.[485] The infant ultimately died after transportation to the Emergency Department and a short PICU course.[486] The findings included bilateral SDH, SAH, and acute rib *277 fractures.[487] While in the PICU, an eye examination revealed retinal hemorrhages throughout both eyes (no normal retina were identified in either eye) with 360° retinal detachment.[488] The authors reported that the child had not suffered AHT and that "choking, vomiting, or paroxysmal coughing (e.g., pertussis) may also result in SDH and RH."[489] The authors concluded that the SDHs and RHs are "consistent with the history of infantile dysphagic choking as consistently provided by the caretaker."[490]

With regard to neck injury, a recent paper by Matshes et al. set forth the hypothesis that hypoxia was indeed the underlying cause for SDHs and RHs, but that neck injury, as opposed to choking or coughing, was the preceding cause of the hypoxia. [491] In that paper, Matshes et al. reported on thirty-five non-sequential infant fatalities from three different medical examiners' offices. Twelve (34%) of these were "confirmed or suspected by history and circumstance to have been subjected to hyperextension and hyperflexion forces." [492] The authors did not describe how the infants were selected or how the neck injury was determined. These twelve hyperflexion/hyperextension cases were compared with twenty-three control infants in whom neck hyperextension/hyperflexion was not identified or suspected. [493] The authors reported that all twelve hyperextension/hyperflexion cases had nerve root hemorrhage (bleeding where the nerves of the neck enter the spinal cord), while only one of the twenty-three control infants had nerve root hemorrhage. [494] The authors indicated that the nerve root hemorrhage in the "shaking injury" infants was evidence that they sustained neck injuries that ***278** interrupted the regulation of breathing, resulting in hypoxia. [495] They reported that all twelve "shaking injury" cases had SDH, while only two of the twenty-three control infants had SDH. [496] This was a statistically significant relationship comparing SDH amongst those with shaking as compared with those without shaking (chi square, p=0.000). The authors concluded that the twelve "shaking injury" cases had neck injuries that resulted in hypoxia and a subsequent SDH. [497]

D. Scientific Critique of the Adjunct Hypotheses

While Geddes and Talbert's computer model is interesting and, perhaps, hypothesis generating, no clinical or physiological data exist to support it. Geddes and Talbert themselves admit that the values utilized in their model are calculations and have not been demonstrated to be true. [498] The authors state that although their model demonstrates the pressure within the vessels surpasses the failure threshold, "[n]o research specifically addressing the question of stress failure of intracranial veins appears to have been reported in the literature." [499] Furthermore, since its publication six years ago, there has been no subsequent confirmation or supporting research published. Finally, given the ubiquity of infant gastroesophageal reflux (spitting up), if choking or gagging on formula were truly a meaningful cause of death, SDH, or RH, would it not already have been identified and published in medical treatises and literature? Yet, this is not the case.

With regard to the Barnes et al. case report, there are several ethical concerns with this case report that impact the scientific validity of the data presented. First, the "case report" bears striking similarities to a case (Zavian Thomas v. State of Texas) [500] in which each ***279** of the co-authors were expert defense witnesses (a disclosure that was not made when published in the medical literature). Second, when confronted in the medical literature with the similarities between this "case report" and that case, [501] the authors chose not to clarify this issue. [502] Finally, there is concern that the "case report" did not present complete clinical information when published in the medical literature. [503]

One condition Talbert himself highlighted as an example of how choking or coughing, in the face of hypoxia, could cause SDH and RH is pertussis (i.e., whooping cough). [504] Pertussis is an airway infection by the bacteria Bordetella pertussis which produces a toxin that causes extensive lung inflammation. [505] Prior to the advent of an effective vaccination for the bacteria, there were over 200,000 cases annually in the U.S., mostly in infants and children. [506] Tens of thousands of deaths occurred each year in the U.S., with over three-quarters of deaths being children younger than two years. [507]

Despite the overwhelming burden of this disease, there have been only two cases of pertussis-associated SDH in over 100 years of published medical literature. The first, from 1885, [508] was a two-year old who died from reported pertussis. The second case was reported ***280** in the American Academy of Pediatrics Red Book (infectious diseases manual). [509] The latter involved

a four-week old from 1968 who had pertussis and developed Staph aureus pneumonia.[510] The infant died as a complication of pneumonia brought on by pertussis.[511]

As with gastroesophageal reflux, given the extensive history of pertussis, with hundreds of thousands of deaths in infants and children, if SDH or RH were a notable feature, would they not already be identified and reported in the medical literature? Yet, this is also not the case. Furthermore, in a well-designed prospective study, conducted over a four-year period, Curcoy et al. examined the eyes of thirty-five infants and young children admitted to their hospital with pertussis.[512] They found none with retinal hemorrhages. Similar findings were repeated in a prospective study by Goldman et al.[513] Finally, Herr et al. prospectively examined the eyes of 100 infants admitted to the Children's Hospital of Pittsburgh with forceful vomiting caused by pyloric stenosis.[514] They also found none with retinal hemorrhages. It appears that there are little scientific data to support the hypothesis that coughing or choking causes SDH or RH.

With regard to the hypothesis of neck injury as a putative cause of SDHs or RHs, beyond the small numbers in the Matshes study, this paper has some significant methodological flaws that make meaningful interpretation impossible. The authors were not blinded to the neck findings, clinical information, or the pathologic findings. How neck hyperextension/hyperflexion was determined was not ***281** described.[515] As neck hyperextension/hyperflexion was a key determinant in whether the infant was a case or control, it needed to be explained how it was actually determined. Finally, and very interestingly, four of the twelve (33%) cases of shaking injury did not have evidence of hypoxic encephalopathy yet still had SDH.[516] Clearly, Matshes' data more strongly supported shaking alone as the cause of SDH, rather than hypoxia.

E. Conclusion

From the clinical perspective, hypoxia is an important consideration. Hypoxia likely plays a role in some of the significant neuro-devastation seen in infants and children who are victims of any traumatic brain injury, not just AHT. However, when it comes to hypoxia causing SDH and RH, there simply is no clinical data or compelling research that supports this contention. If examined on the Oxford Centre for Evidence-Based Medicine rating scale,[517] the current level of evidence on the topic is of the lower kind--level 4. Comparatively, the level of evidence arguing against this hypothesis is stronger--level 2b.

VII. The Daubert Analysis

The determination of what is "reliable" expert testimony is a problem that has vexed jurists and legal scholars for hundreds of years. In one of the earlier historical legal writings on the subject matter, one eminent jurist and legal scholar, Judge Learned Hand, wrote:

> Having briefly considered the history of the present position of expert witnesses, the really practical question is whether it is the best way to use the information they can give. There are two things I wish to prove: first, that logically the expert is an anomaly; second, that from the legal ***282** anomaly serious practical difficulties arise.[518]

Although Judge Hand offered his brethren of the long robe a solution to the predicament of the expert witness,[519] this legal malady continues to ail jurisprudence to this very day.

For scores, the common-law rule governing admissibility of scientific expert testimony was the Frye standard (or the "general acceptance" test).[520] With the enactment of the Federal Rules of Evidence (FRE) in 1975 and the Daubert court's subsequent countenance of them in many jurisdictions, the Frye standard came to pass.[521] FRE 702 states that expert witness testimony

shall be restricted to "scientific, technical, or other specialized knowledge" that is the "product of reliable principles and methodology." [522] The Daubert court interpreted the adjective "scientific" to mean "a grounding in the methods and procedures of science" [523] and tethered "evidentiary reliability" to "scientific validity." [524] From there, the Daubert court enunciated its renowned checklist of factors for assessing "scientific validity." [525] Subsequent elaboration on the limits and meanings of FRE 702 in Joiner [526] and Kumho [527] have clarified that ***283** the expert's methodology cannot be based solely upon "the ipse dixit of the expert" [528] and must be "properly applied" to the particular "facts of the case." [529]

In child abuse cases, the gatekeeper is confronted with primarily [530] three legal issues:

1) Does a physician's testimony constitute "scientific," "technical," or "other specialized knowledge?"

2) Is the physician's testimony the "product of reliable principles and methodology?"

3) Has the physician reliably applied those principles and methodology to the particular facts of the case?

The first and second issues are generally addressable here; the third requires specific application to specific cases with specific fact patterns.

A. Does a Physician's Testimony in Child Abuse Cases Constitute "Scientific," "Technical," or "Other Specialized Knowledge?"

Although not the dispositive issue, it is not a superfluous matter to determine whether a physician's testimony in child abuse cases constitutes "scientific," "technical," or "other specialized knowledge." Many legal scholars have noted that the adjective "scientific" connotes a greater reliability and even an "aura of infallibility." [531] Although ***284** empiric data from civil and criminal juries have not supported that "aura of infallibility" concern, empiric data have confirmed increased levels of juror attention to, critique of, and reliance upon appropriately presented scientific information. [532]

One distinguished evidentiary scholar has asserted that physician testimony in support of shaken baby syndrome is "non-scientific," stating that it is premised upon primarily "anecdotal" evidence. [533] That learned scholar goes on to assert that physician testimony against shaken baby syndrome is "scientific," arguing that it attains "empiric" validity from Duhaime's biomechanical study in 1987. [534] Although the learned scholar makes no clarification as to what constitutes "anecdotal" evidence or whether there is evidence other than "anecdotal" evidence in support of shaken baby syndrome, he still concedes its admissibility under Daubert scrutiny. [535] The shortcomings of this "scientific" /"non-scientific" analysis will be discussed in further detail below.

In defining "scientific," the Daubert court stated that "the adjective 'scientific' implies a grounding in the methods and procedures of science." [536] Generally speaking, a physician's testimony has been and is considered "scientific." A physician's education includes the scientific basis of health and disease. [537] Rooted in a foundation of core science subjects, such as biology, physics, chemistry, and biochemistry, physicians are trained to apply those scientific ***285** principles to the human body to better understand the physiology of the body and the pathology of disease that can affect it. Physicians are further trained to use scientific literature to compare alternative approaches to diagnosis and treatment. [538] In addition, physicians receive basic training on statistical analysis, often applying those principles to critically evaluate the medical literature.

Courts that have confronted the issue have commented, "[c]linical diagnoses bear the marks of science." [539] As the American Medical Association stated in its amicus brief in Daubert:

> 'Scientific knowledge' is that body of knowledge that has been learned or developed in accordance with rigorous scientific methodology. The scientific method involves replicable, empirical testing of hypotheses . . . . Medical knowledge is one kind of scientific knowledge. It is acquired through the application of the scientific method to questions concerning the effects of various interventions on human health. [540]

In fact, after considering whether the expert testimony of the physicians and biostatisticians involved in the case was "scientific, technical, or other specialized knowledge," the Daubert court concluded that its analysis was "limited to the scientific context because that is the nature of the expertise offered here." [541] Additionally, some courts and scholars have made a distinction between "hard" and "soft" sciences. [542] Because this distinction has no true correlation with reliability, we will not delve into further discussion of this issue.

The natural extension of this analysis is to consider whether there are any features or characteristics of a physician's methodology in AHT/SBS cases that make it less scientific or non-scientific. The simple answer is no. The physician in AHT/SBS cases employs no ***286** different methodology than the ER physician who assesses life or death scenarios in the emergency room, or than the neurosurgeon who assesses the cause and treatment of intracranial bleeds, or than the forensic pathologist who assesses the cause and manner of death in a variety of cases. All employ the "differential diagnosis" methodology, a methodology rooted in the scientific method. [543]

Furthermore, if, as the Daubert court stated, the "scientific method" is a "process" of "generating hypotheses and testing them to see if they can be falsified," [544] then it is without question that AHT/SBS has been subjected to the "scientific method." As Narang pointed out in his prior article, physicians from all across the world have not only tested AHT/SBS from a variety of different perspectives but in a variety of different disciplines: biomechanics, pathology, radiology, ophthalmology, neurosurgery, and general pediatrics. [545] In its recent assessment of the forensic sciences, the National Research Council stated:

> Scientists continually observe, test, and modify the body of knowledge. Rather than claiming absolute truth, science approaches truth either through breakthrough discoveries or incrementally by testing theories repeatedly. [546]

In fact, it is this very scientific methodology--this process for seeking greater scientific precision--that has prompted physicians to modify constrictive terminology, such as "shaken baby syndrome," to more inclusive terminology, such as "abusive head trauma." However, as child abuse physicians have utilized the science to be more precise, they have ironically been criticized for being "less scientific." ***287** [547]

Finally, the assertion that the evidence basis in support of AHT/SBS is "primarily anecdotal" is not only factually inaccurate but logically overly simplistic. "Anecdotal evidence" refers to evidence from anecdote, or more simply speaking, evidence that is primarily based upon personal experience and not subjected to the rigors of scientific analysis and scrutiny. [548] While certainly some evidence of shaking has been "anecdotal" (i.e., some admissions of shaking by caretakers to physicians), there are other forms of scientific evidence that support the "shaking" proposition. Recently, animal studies (on species that have highly similar head and neck anatomic structures to the human infant) have reproduced the very injuries--SDHs and RHs--reported to be found as a result of shaking. [549] And as detailed above, there are biomechanical studies that support the "shaking" proposition. [550] More importantly, shaking accounts of perpetrators have not been merely anecdotally asserted and accepted but have been subjected to various aspects of scientific scrutiny, from examination of the specifics and repetitiveness of shaking events [551] to the correlation of those accounts to the presence or absence of physical trauma signs upon the body. [552]

However, and most importantly, the evidence for or against "shaking" is not merely a biomechanical question. The analysis does not simply end at the question of whether the estimated forces required to cause *288 SDHs have or have not been reproduced in biomechanical studies; there are clinical questions as well. Assuming, arguendo, that impact with a surface (soft or hard) is required to reach the estimated force thresholds of SDHs, then what? Because it is already well-established that many abusive head injuries do occur with impact against a soft surface, what next? If valid probabilistic judgments are to be made, further clinical questions must be answered--such as: With what frequency/commonality do the associative findings (SDHs, RHs, fractures, etc.) occur? What degree of reliability is there in those findings? How common are those findings in low impact/simple short fall events? And are there other clinical variables that are strongly indicative of accidental or abusive events? The focus on solely the biomechanical question is itself a prime example of anchoring bias. [553] It is the assimilation and assessment of all types of evidence (as will be discussed in further detail herein below), not just biomechanical data, which leads to the most reasonable conclusion on this matter.

Therefore, physician testimony in AHT/SBS cases is, and should be, considered "scientific."

B. Is the Physician's Testimony in AHT/SBS Cases the "Product of Reliable Principles and Methodology?"

As mentioned above, determining what is "reliable methodology" in an expertise or a subject matter that is completely foreign to one's own is no simple task for anyone. Although the Daubert court listed a checklist of factors to consider in assessing "scientific reliability," some courts have questioned the hard-and-fast application of those factors to clinical medicine. [554] While not a *289 perfect fit to clinical medicine, the Daubert factors are malleable to clinical medicine and help in the overall analysis of reliability.

As the Daubert court ultimately stated, it is the "principles and methodology," not the "conclusions they generate," that are of paramount importance. [555] As Narang pointed out in his first part of this analysis, the methodology physicians employ in coming to the diagnosis of AHT is no different from the methodology physicians employ in arriving at any medical diagnosis--it is the differential diagnosis methodology. [556]

Legal scholars have expounded that, while the derivation of scientific principles involves inductive reasoning, in court, the explanation of the methodology follows a deductive, syllogistic format:

> Although scientific propositions are derived inductively, in the courtroom scientific testimony is ordinarily presented in a deductive, syllogistic format . . . . The major premise is a principle, procedure, or explanatory theory derived by the inductive, scientific technique. The physician applies that major premise to the facts of the case, namely, plaintiff's case history. The symptoms displayed by this specific plaintiff are the witness's minor premise. That case history might show that plaintiff has experienced symptoms A, B, and C. The result of applying the major to the minor premise is a conclusion, the witness's opinion on the merits of the case . . . . Hence, the 'path to the witness's final opinion' leads through the major and minor premises on which the expert relies. [557]

Applying such to AHT/SBS cases, the "major premise" would be the scientific principles and evidence underlying the AHT/SBS diagnosis. The "minor premise" would be the utilization of the differential diagnosis methodology to the specific facts of the AHT/SBS case.

Thus, the analytic journey from here courses through three paths: 1) determining whether the scientific principles and evidence underlying the AHT/SBS diagnosis are in fact reliable, i.e., the "major *290 premise"; 2) defining what exactly the "differential diagnosis methodology" is; and 3) exploring whether the "differential diagnosis methodology" is, in general, a reliable methodology for applying the major premise to the specific facts of a case, i.e., the "minor premise." The determination of whether a physician has validly applied the differential diagnosis methodology to a specific fact pattern is a mental endeavor a

particular gatekeeper must endure. Toward the conclusion of this article, we will discuss some tools available to the gatekeeper for confronting this challenge.

1. Are the scientific principles and evidence underlying the AHT/SBS diagnosis "reliable" (i.e., the "major premise")?

The Honorable Justice Stephen Breyer commented:
The search is not a search for scientific precision. We cannot hope to investigate all the subtleties that characterize good scientific work. A judge is not a scientist, and a courtroom is not a scientific laboratory. But consider the remark made by the physicist Wolfgang Pauli. After a colleague asked whether a certain scientific paper was wrong, Pauli replied, 'That paper isn't even good enough to be wrong!' Our objective is to avoid legal decisions that reflect that paper's so-called science. The law must seek decisions that fall within the boundaries of scientifically sound knowledge. [558]

Justice Breyer's statements are not merely colorful commentary or interesting narrative. They express the careful balance that is sought between two competing evidentiary goals--scientific soundness and a liberal, flexible approach to admissibility. They are notable in light of recent court decisions employing stricter standards of admissibility [559] and judicial surveys expressing confusion regarding ***291** the proper criteria for admissibility. [560] And they are especially noteworthy because some legal scholars have fallaciously confounded standards for diagnostic sufficiency with standards for criminal conviction sufficiency. [561]

So the question is simple: Is the science underlying AHT/SBS "junk science," or science "that's not even good enough to be wrong?" One legal scholar made the following assertions that formulated the premise of Narang's first article:

1) That "the scientific underpinnings of SBS have crumbled over the past decade"; [562]

2) That, "as evidence-based medicine . . . required doctors to derive their research from methods that are scientific and statistically rigorous," doctors learned that the diagnosis was predicated upon "flawed science"; [563] and

3) That "as technology and scientific methodology advanced, researchers questioning the basis for SBS reached a critical mass" [564] (i.e., no longer a "general acceptance").

These assertions had been "reified" in prior [565] and subsequent legal commentary, [566] and unfortunately, found scientifically unscrutinizing ***292** ears in three Supreme Court justices in the recent Cavazos v. Smith decision. [567] Simply stated, these assertions question the reliability of the scientific principles that formulate the physician's "major premise" in the syllogistic argument.

In response to those assertions, in the first part of this analysis, Narang examined the scientific underpinnings of two findings commonly seen in AHT/SBS--SDHs and RHs. Narang examined the science supporting the association of these findings with AHT/SBS. As evidence of the reliability of those associative findings, Narang referenced over 200 evidence-based, scientific studies (not editorials or reviews--but clinical studies), detailed the scientific validity of fifteen of those articles, and subjected them to the four Daubert factors used in assessing scientific reliability. [568] In conclusion, Narang determined that not only did this scientific literature meet Daubert criteria for reliability but that there was no "critical mass" questioning the diagnosis or change in general acceptance of the diagnosis. [569]

Interestingly, having now been confronted with promulgation of that scientific literature, those same scholars have shifted their arguments. [570] Whereas before they argued that there was little-to-no evidence-based medical literature supporting the

diagnosis (likening it to an "inverted pyramid with a small database"), [571] they have now posited that Narang's use of the "voluminous" scientific literature "serves to intimidate those who are not familiar with its methodological shortcomings." [572] Whereas before they alleged that the AHT/SBS literature did not engage in the scientific or statistical rigors required by evidence-based medicine, [573] now they state that *293 the literature either misstates the significance of the P-value or does not sufficiently calculate the strength of statistical associations by providing posterior probabilities. [574] And whereas before they asserted that "researchers questioning the basis for SBS reached a critical mass," [575] now they argue either that "it is increasingly difficult to gauge the extent to which doctors in general agree" [576] or that it is "insufficient to rely on the fact that some professional groups accept or endorse the diagnosis of SBS/AHT." [577]

While it certainly would be entertaining to engage in ongoing debate about "prosecutors' fallacies," "improper classifications," and "shifting paradigms," such an endeavor would be neither productive nor relevant. Ultimately, at this point in the analysis, the focus must be on whether the scientific principles forming the basis of the AHT diagnosis are reliable, i.e., whether there is reliability in the physician's major premise of the syllogistic argument. It is important to note at this point that it is simply beyond the scope of this article to state ALL the scientific principles a physician utilizes in arriving at the AHT/SBS diagnosis. For example, there are many developmental principles of infants and children that physicians utilize in correlation with a history in assessing the reliability of that history. Additionally, there are principles derived from various studies investigating alternative causes of SDHs, RHs, fractures, and bruises that physicians utilize in determining whether to rule out other potential causes of those findings. [578]

In his first article, Dr. Narang attempted to highlight some of the key scientific principles regarding SDHs and RHs. However, despite repetition, [579] this still resulted in confusion by some scholars [580] about *294 what exactly those scientific principles were. Thus, given the importance of those principles to the overall analysis, we will restate those scientific principles below (along with their evidence-based medicine levels) in bold, so as to hopefully avoid any further confusion:

1) That trauma is the most common cause of SDHs--based upon epidemiologic studies in young children, both prospective and retrospective, from multiple countries (level 3b evidence); [581]

2) That when examining the breakdown of trauma-caused SDHs (i.e., accidental v. non-accidental), non-accidental trauma is by far more common--based upon epidemiology and pathology studies in young children, both prospective and retrospective (level 2b evidence); [582]

3) That SDHs being much more common in non-accidental trauma than in accidental trauma is a statistically significant conclusion reached by numerous well-designed, prospective clinical studies (level 2b evidence); [583]

*295 4) That severe RHs being much more common in non-accidental trauma than in accidental trauma is a statistically significant conclusion reached by numerous well-designed, prospective clinical studies (level 2b evidence); [584]

5) That severe RHs carry a high specificity and positive predictive value for non-accidental trauma--based upon prospective, validating clinical studies and systematic reviews (level 1b and 2a evidence); [585]

6) That the absence of a trauma history, in the presence of traumatic injuries, holds a high specificity and positive predictive value for non-accidental trauma--based upon several well-designed, prospective clinical studies (level 2b evidence). [586]

As Narang detailed in his first article, these scientific principles are the result of methodologies specifically designed to minimize *296 circularity and bias. [587] They produce results that have been repeatedly reproduced by other physicians. [588] And they

satisfy Daubert criteria for reliability. [589] Findley et al. responded by criticizing the validity of the literature on essentially two grounds--(1) that the methodology of "virtually all" of the studies is marred by "circularity" [590] and (2) that there are various errors clinicians have made in interpreting the results of these studies. [591] As "circular" methodology more directly pertains to the major premise (i.e., the scientific principles), it will be discussed below. As "interpretive errors" pertain more to the minor premise (i.e., the application of those principles to the particular facts of a case), they will be addressed in the "minor premise" section below.

Since "circularity" has been alleged to infect "virtually all" of the literature, prior to addressing it, it is appropriate to identify the scientific principles that are deducible from the Accidents, Bleeding Disorders, Biomechanics, and Hypoxia sections discussed above. These scientific conclusions are:

1) That short falls occurring in objective settings, such as hospitals, have not resulted in subdural hematoma or death--based upon several consecutive case series (level 3b evidence); [592]

2) That severe injuries or death resulting from short falls are rare events-- based upon well-designed, prospective studies and systematic reviews (level 2a evidence); [593]

3) That certain clinical variables, such as apnea and severe RHs, demonstrate high positive predictive values for non-accidental trauma based upon prospective, validating ***297** clinical studies and systematic reviews (level 1b and 2a evidence); [594]

4) That most bleeding disorders are rare, the more common bleeding disorders typically are mild, and intracranial hemorrhage resulting from bleeding disorders is a rare complication of the more severe rarer diseases--based upon clinical studies (level 3b evidence; level 1b symptom prevalence evidence); [595]

5) That biomechanical studies have shown mixed results as to whether shaking can result in the estimated mechanical forces needed to cause SDHs; [596]

6) That biomechanical studies have shown that RHs can result from shaking; [597]

7) That biomechanical studies have NOT shown that neck "failure" must result prior to the estimated forces required for SDHs being achieved; [598]

8) That macroscopic SDHs are not associated with hypoxia--based upon several well-designed radiology and pathology studies (level 2b evidence); [599]

9) That severe RHs are not associated with hypoxia--based upon well-designed clinical studies and animal studies (level 2b evidence); [600] and

***298** 10) That adjunct hypotheses of hypoxia (such as "dysphagia/choking," "coughing," or "dural immature vascular plexus") resulting in SDHs and/or RHs are supported by the lowest levels of evidence-based medicine (level 4 or 5), whereas evidence against such hypotheses is much stronger (level 2b). [601]

As with the principles Narang discussed in his first article, and as demonstrated in the sections above, these scientific principles have been subjected to "falsifiability." They have been peer reviewed and published. They represent the highest levels of statistical analysis. Their results have been reproduced in multiple studies and across various lines of research. And finally, as

these principles form the basis of the generally accepted diagnosis of AHT/SBS, [602] they are generally accepted. Consequently, they are also valid and surpass Daubert scrutiny.

With regard to the "circularity" concerns raised by Findley et al., [603] interestingly, much like some of the other positions, this "circularity" argument has also shifted. Whereas before Findley et al. argued that the "circularity" consisted of including SDHs and/or RHs in the defining inclusion criteria of a study, [604] now they argue that "major trauma" is the assumed circular premise in "virtually all" of the literature. [605]

***299** Narang addressed this "circularity" [606] argument in his first article. [607] His retorts--of why "circularity" does not explain the historical articles (articles that initially identified these associative injuries prior to the designation of a syndrome) or why NO scientific studies have consequently been created that are not circular and show a lack of this association--have yet to be addressed. Setting aside these unrequited responses, there are several other fatal shortcomings to this argument.

First, as scientists, physicians are always asking how to test a hypothesis soundly. Well before the admonishments of Findley et al., physicians themselves recognized the methodological problems of circularity in some of the literature. [608] In efforts to remedy these flaws, physicians created various a priori definitions for AHT (that excluded SDHs and RHs) [609] and even limited some studies to simple comparative cohorts of witnessed accidents versus judicially confessed abusive acts. [610] Yet, Findley et al. still find these to be "circular." If that is the case, then the simple question is: What would be "non-circular methodology?" If confessions are circular or invalid and all a priori definitions presume something, then what would be a satisfactory methodology for a study? What is the sound methodology Findley et al. would utilize in conducting a study? This is a ***300** simple question that Findley et al. have yet to answer. It appears that what is guised as a "circular" critique is actually a philosophical and logical quagmire that devolves into the answer that "nothing," then, could be non-circular.

Second, do doctors really assume that SDHs are caused by major trauma? Or is that not a proven entity? Closer scrutiny of the "circularity" label reveals that the study methodologies are not actually "circular" but simply systematically deductive. Narang spent considerable time in his first article detailing the historical progression of the understanding of SDHs from an infectious etiology to a traumatic etiology. [611] And multiple studies have not only validated that premise [612] but determined that trauma is actually the leading cause of SDHs in infants and children. [613] From years of research, physicians have compiled a list of additional potential causes of SDHs. [614] If one is to then study a particular subset (such as accidental trauma versus non-accidental trauma) of those causes, is not the only logical process to then attempt to exclude all other potential causes prior to studying that subset? This is the methodology that physicians have consistently employed in the various studies discussed above. And this is not "circular"; it is systematic and deductive.

Finally, Findley et al.'s critique of circularity itself suffers from a logical fallacy--the fallacy of hasty generalization. Findley et al. wish to place hundreds of studies into a box. [615] And they wish to paint, with one broad swath, a "CIRCULAR" sign along the side of that box, and set that box aside. [616] But are those studies, in fact, circular? For ***301** example, Chadwick's systematic review of short falls is not circular because a history of a short fall is not a criterion used to determine that a child has died. [617] Multiple studies of short falls avoid circularity by including EVERY fall seen at the respective institution in a given period of time and by not explicitly separating the children into groups of abused and non-abused children. [618] These studies are manifestly non-circular because the measurement of the outcome variable does not even consider the predictor variable. Finnie's study made circularity impossible by randomly assigning specific lambs to be injured or not injured. [619] Multiple anthropometric and FEM studies use experimental designs that are simply not circular. [620] And the Odom study of the prevalence of RHs in the setting of chest compressions avoided circularity by considering only children thought not to be abused prior to their eye exam. [621] These are but a few of the examples.

We are not interested in the elementary back-and-forth of: "yes, it is circular"; "no, it is not circular." The science speaks for itself. In hopes of transparency, and not "intimidation," [622] we have presented a reasonable sample of that science. The scientific studies that form the basis of the scientific principles discussed above have been laid out for the reader to judge for himself/herself. For countless physicians, these studies and the principles they have produced are scientifically valid. They comprise the "major premise" of the AHT syllogistic argument.

***302** 2. What is the "Differential Diagnosis Methodology?"

Stedman's Medical Dictionary defines "differential diagnosis" as "the determination of which of two or more diseases with similar symptoms is the one from which the patient is suffering, by a systematic comparison and contrasting of the clinical findings." [623] Simply stated, from a medical perspective, it is the list of diseases that physicians consider as possible causes for the signs or symptoms from which the patient is suffering. [624] Interestingly, however, from a legal perspective, courts have varied in their interpretation and understanding of it. Some courts have interpreted it to be the methodology for arriving at causation by "ruling out" alternative causes. [625] Others have interpreted it to require both a "ruling out" and "ruling in" process for arriving at causation. [626] And others have even created legal concepts, such as "differential etiology," declaring it to be different from "differential diagnosis." [627]

While courts have expressed variable levels of understanding of the differential diagnosis methodology, it is cognitive scientists who have provided the deepest and richest understanding of that ***303** methodology. Decades of research has revealed that, whereas it was once thought that physician clinical reasoning proceeded in a discretely linear fashion known as Bayesian analysis, [628] the diagnostic process is actually a nonlinear, unstructured method of problem solving that employs both inferential and deductive reasoning. [629] On occasion, and for various reasons (such as clinical exigency), physicians may bypass the hypothetico-deductive approach and utilize a different reasoning process known as "heuristics," or problem-solving shortcuts. [630]

In the differential diagnosis methodology, the physician gathers historical information on a patient's symptoms and signs and generates hypotheses (a.k.a., the differential diagnosis). [631] Through the attainment of additional clinical information (via various diagnostic tests), the physician goes through an inferential and deductive process of hypothesis refinement until a consistent "working diagnosis" is achieved. [632] Hypothesis refinement utilizes a variety of reasoning strategies--probabilistic, causal, and deterministic--to discriminate ***304** among the existing diagnoses of the differential diagnosis. [633] While being mindful of the pitfalls of heuristics, the physician ultimately proceeds to hypothesis confirmation when the laws of diagnostic "adequacy," "coherency," and "parsimony" are satisfied. [634] In the simplest sense, the methodology relies on process-of-elimination reasoning. As one eminent evidentiary scholar stated, "[i]n differential diagnosis, if there are four possible diagnoses and you eliminate three, logic points to the last illness as the correct diagnosis." [635]

In AHT/SBS cases, the differential diagnosis depends on the findings presented. Soft tissue injuries (such as bruises) have a differential diagnosis. Fractures (either long bone or skull) have a differential diagnosis. And intracranial findings (such as SDHs or cerebral edema) and ophthalmologic findings (such as RHs) also have a differential diagnosis. [636] It is the physician's task to parse through the historical information, the physical examination, and the laboratory and radiologic results to arrive at a unifying diagnosis that satisfies the criteria of "adequacy," "parsimony," and "coherency." As Narang has already stated, in many cases but obviously not all, ***305** the unifying diagnosis will be trauma. [637] From there, the physician will again utilize the historical information, the physical examination, the laboratory/radiology results, the medical literature, and his/her experience to distinguish between accidental and non-accidental trauma. This process/methodology will be examined in further detail below.

3. Is the "differential diagnosis methodology," in general, a reliable methodology for applying the major premise to the specific facts of a case (i.e., the "minor premise")?

A scholar once mused that both Sherlock Holmes and Captain Spock agreed on the same proposition"when you have eliminated the [other possibilities], whatever remains, however improbable, must be the truth." [638] That scholar went on to state, "when the most logical human and a Vulcan agree on a proposition, that proposition must have merit." [639] While this is not the crux of our analysis, it is an interesting starting point.

Courts have long held that the differential diagnosis methodology is a reliable methodology for arriving at specific causation in tort cases. [640] In those cases, courts have also commented that "[d]ifferential diagnosis is a well-recognized and widely used technique in the medical community to identify and isolate causes of disease and death." [641] In criminal cases, and more specifically in AHT/SBS cases, the courts have concluded no differently. [642] However, judicial decisions ***306** have offered little insight into why the methodology is reliable. Additionally, recent investigation into the forensic sciences has raised concern over implicit contextual and cognitive biases in the methodologies underlying forensic judgments. [643]

Thus, these concerns may justify a re-evaluation of previously unchallenged methodologies. With all due respect to the acumen of the brethren in the long black robe, the ipse dixit of the judicial expert may be no better than the ipse dixit of the medical expert. So then, what exactly is it that makes the differential diagnosis methodology reliable? And has it been unsalvageably infected with the contextual and cognitive biases that seem to undermine other forensic judgments?

One oft-asserted validation of the differential diagnosis methodology is that it is a methodology employed for making life-or-death decisions and, ergo, must be reliable. The Advisory Committee Notes for the drafters of the Federal Rules of Evidence indicates that one of the drafters' objectives was to formulate evidentiary standards in accord with experts' practices in the field. [644] Those Notes asserted that if a physician "in his own practice" considers a certain type of data and "makes life-and-death decisions in reliance on them," common ***307** sense suggests that reasoning relying on such data should also be acceptable in the courtroom. [645] Those same advisory committee comments were echoed almost a decade after the Federal Rules of Evidence went into effect by a leading evidentiary commentator, Professor Charles Nesson, when he published a celebrated article arguing that testimony based on the reasoning processes commonly used by medical diagnosticians ought to be admissible in court. [646]

Another factor pointing to its reliability is that the differential diagnosis methodology is rooted in the scientific method. If the core of science is "falsifiability"--the formulation of hypotheses and the conduct of systematic experimentation or observation to validate or "falsify" those hypotheses--then the differential diagnosis methodology is science embodied (no pun intended). Physicians formulate causal hypotheses for illness and other medical injuries and utilize the differential diagnosis methodology for the systematic validation or falsification of those hypotheses. But not only has the differential diagnosis employed "falsifiability," it has been subjected to it as well. As mentioned above, cognitive scientists have conducted years of research on the methodology to assess and identify its strengths and weaknesses. In that course, the differential diagnosis methodology has itself been subject to peer-reviewed publication. [647]

Additionally, the methodology's process-of-elimination reasoning is utilized not just by physicians, but by lay persons and lawyers as well. [648] It is not just "generally accepted"; it is "generally utilized." As Karl Popper, one of the preeminent philosophers of science, stated, science is only "common-sense knowledge writ large." [649]

The most reasonable challenge to the reliability of the differential ***308** diagnosis methodology (or to any methodology that involves human analytic reasoning) is bias and errors of cognition. Findley et al. have labeled some of these as "observer bias" and "interpretive errors" (such as "improper classifications" and "the prosecutor's fallacy"). [650] The National Research Council, in its recent global assessment of the forensic sciences, generally described them as "cognitive" and "contextual" biases. [651]

The Council went on to warn that "[t]he traps created by such biases can be very subtle, and typically one is not aware that his or her judgment is being affected." [652] With regard to the forensic disciplines, the Council concluded that:

> Unfortunately, at least to date, there is no good evidence to indicate that the forensic science community has made a sufficient effort to address the bias issue; thus, it is impossible for the committee to fully assess the magnitude of the problem. [653]

Thus, the Council recommended that the National Institute of Forensic Science fund and conduct further research on human observer bias and sources of human error in forensic examinations. [654]

In its discussion of error rates, the Council identified four statistical principles that are especially helpful in assessing error rates: sensitivity, specificity, positive predictive value, and negative predictive value. [655] The Council stated that a "global error rate" can be estimated by summing the percentage of false positives and false negatives of a particular test. [656] For example, a test with a 95% sensitivity has a 5% false negative rate. [657] And a test with a 97% specificity ***309** has a 3% false positive rate. [658] Thus, the "global error rate" of that particular test could be estimated as four percent [(5+3)/200 x 100 = 4%]. While a bit simplistic, and not dispositive of the reliability of a particular test, it does offer some statistical quantification of the "error rate" criteria sought by Daubert. Narang discussed the application of these statistical principles (and odds ratios) to the SDH/RH literature in his first article. [659] The application of these statistical principles in the differential diagnosis methodology of a child abuse case will be exemplified below.

As mentioned above, cognitive scientists have provided invaluable insight on bias in decision making, specifically in clinical medicine. In Daniel Kahneman's Nobel prize acceptance essay, Maps of Bounded Rationality: A Perspective on Intuitive Judgment and Choice, the author summarizes a long and an arduous journey wherein he and his colleague, Amos Tversky, "explored the psychology of intuitive beliefs and choices and examined their bounded rationality." [660] Kahneman and Tversky identified "a two-system view" that distinguishes intuition from reasoning. [661] "The operations of System 1 [intuition] are fast, automatic, effortless, associative, and difficult to control or modify." [662] "The operations of System 2 [[reasoning] are slower, serial, effortful, and deliberately controlled; they are also relatively flexible and potentially rule-governed." [663] "A defining property of intuitive thoughts is that they come to mind ***310** spontaneously, like percepts," and can result in rash judgments, also known as "heuristics." [664] This concept of "heuristics" has been a vital concept in understanding the pitfalls of clinical decision making. [665]

The concept of "judgment heuristics" informs us that "intuitive judgments of probability are mediated by attributes such as similarity and associative fluency" and "are not intrinsically related to uncertainty." [666] Other attributes of heuristic judgments include susceptibility to availability bias, accessibility bias, anchoring, representativeness, overweighting, and attribute substitution, to name a few. [667] Kahneman notes that "people rely on a limited number of heuristic principles which reduce the complex tasks of assessing probabilities and predicting values to simpler judgmental operations." [668] While these heuristics can be quite useful, "sometimes they lead to severe and systematic errors." [669]

However, Kahneman and Tversky determined that errors of heuristic judgments can be tempered by the slow, serial, effortful, deliberate, and rule-oriented operations of System 2. [670] The efficacy of System 2, in its ability to mitigate heuristic judgments, is impaired "by time pressure," "by concurrent involvement in a different cognitive task," and even "by being in a good mood." [671] The authors state:

> The central finding in studies of intuitive decisions, as described by Klein (1998), is that experienced decision makers working under pressure, such as captains of firefighting companies, rarely need to choose

> between options because in most cases only a single option *311 comes to their mind . . . . Doubt is a phenomenon of System 2, a meta-cognitive appreciation of one's ability to think incompatible thoughts about the same thing.[672]

"Conversely, the facility of System 2 is positively correlated with intelligence, with 'need for cognition,' and with exposure to statistical thinking."[673] Thus, the question to be asked is: How susceptible is the differential diagnosis methodology in child abuse cases to heuristic judgments? It would be scientifically irresponsible to state that the differential diagnosis methodology employed in child abuse cases is immune to heuristic judgments. However, several factors make it less susceptible. First and foremost, the exigency of "time-pressure" circumstances is not one encountered by the child abuse pediatric consultant. Unlike the firefighter, the emergency room physician treating a patient suspected of a myocardial infarction (a.k.a., heart attack) or the intensive care physician running a code, the child abuse pediatric consultant does not experience those time-pressured circumstances. Like other diagnostic consultants, such as infectious disease or endocrinology, the child abuse pediatric consultant has hours to days (if not weeks in certain circumstances) to cogitate upon a differential diagnosis, to order appropriate laboratory and radiology tests, to confer with other subspecialists and interdisciplinary partners, and consequently, to further refine that differential.

Second, the lack of time pressure naturally creates an environment that is suited to a "need for cognition." The child abuse pediatric consultant has ample opportunity and resources for the creation and resolution of doubt. But as Kahneman and Tversky warn, bias is often implicit and goes unrecognized.[674] So what assurance is there that even the cogitating, unpressured child abuse consultant is not shackled with implicit biases?

There are two other important attributes that mitigate the impact of any implicit biases--the multi-disciplinary approach and the utilization of statistical thinking/EBM (evidence-based medicine). *312 Child abuse pediatric consultants often engage in multi-disciplinary evaluations of child abuse cases. These evaluations involve the cognitive efforts of multiple pediatric subspecialists--radiologists, ophthalmologists, neurosurgeons, hematologists, orthopedic surgeons, pathologists, and child abuse pediatricians. Additionally, social interdisciplinary partners (such as law enforcement officials and social workers) provide valuable information that typically is not obtained in routine medical history gathering. Thus, a system is forged whereby comprehensive information is collectively gathered, shared, and evaluated. And as mentioned above, this is not a process amenable or available to all clinical situations and all circumstances (i.e., the ER physician in an emergency situation or the critical care physician in a critical situation). Is this an error proof process? Of course not. Is this an indictment of particular physicians? Of course not. It is merely a recognition of the fact that the comprehensive and collective cognitive operations of the many at least minimize the risk of undetected implicit bias in the single.

Finally, as demonstrated in Narang's first article and in the sections above, physicians have utilized EBM to improve the scientific data in child abuse research. As one cognitive scientist stated: "Evidence-based medicine is the most recent, and by most standards the most successful, effort to date to apply statistical decision theory in clinical medicine."[675]

Physicians have incorporated rigorous design methodologies and statistical analyses (such as logistic regression) to account for confounding variables and bias. These have resulted in not only scientific principles with high degrees of statistical confidence and probability but in principles that have been reproduced, along multiple lines of research, by various physician scientists across the world. As Narang discussed in his first article, the concept of convergent validation offers explanation for their increased validity.[676] Kahneman echoes this in his conclusion: "The claim that cognitive illusions will occur unless they are prevented by System 2 sounds *313 circular, but it is not. Circular inferences are avoidable because the role of System 2 can be independently verified in several ways."[677]

Despite these insulations, is there room for improvement in minimizing the potentiality of implicit bias in clinical decision making? Certainly. Probably the most important one is the need for increased recognition of and education about the topic. [678] As judges have recently increased awareness and education on implicit bias in judicial decision making, physicians need to follow suit. Currently, early medical education involves some "problem-based learning [on] the formulation and testing of clinical hypotheses." [679] But detailed, intensive education about the pitfalls of clinical decision making--i.e., the pitfalls in "hypothesis generation," "diagnostic creation and revision," and "probability estimation and revision"-- is lacking. [680] Thus, increased education in medical school and continuing medical education would be good steps in this direction.

Another consideration in assessing the reliability of the differential diagnosis methodology in AHT/SBS cases is whether it is tethered to the standards of medical practice. Is the same methodology employed in AHT/SBS cases as in other medical cases? Or is it a peculiar diagnostic methodology? In Narang's first article, and herein above, Narang argued that the methodology employed in coming to the diagnosis of AHT is no different from the methodology employed in arriving at any medical diagnosis. [681] Findley et al. have asserted that the medical decision making in AHT/SBS cases differs from other medical diagnoses, such as migraine headaches, because in those diagnoses, unlike in AHT/SBS, "doctors generally correlate the patient's description of the symptoms and their onset (the patient history) with objective medical data (such as lab results) and response to treatment." [682]

*314 This assertion is, in a word, wrong. As has been demonstrated above, physicians do order a host of laboratory and radiologic tests (bleeding studies, bone health labs, x-rays, CTs, MRIs, etc.) and correlate those results with a patient's history. Physicians do effect a treatment--placement in a safer environment--and assess the re-occurrence of any symptoms or injuries in that safer environment. In fact, it is the very inconsistency of the history provided by a caregiver with these objective results that is the cornerstone of the AHT/SBS diagnosis. So the more pertinent question is whether there are other medical diagnoses where there is inconsistency between history and objective medical data and the differential diagnosis methodology is also employed in arriving at that diagnosis.

The answer is, in a word, yes. There are multiple diagnoses that fit this bill--pediatric condition falsification, anorexia nervosa, and drug seeking behavior, to name a few. However, a detailed analysis of one should crystalize this point. The diagnosis of bulimia nervosa is "binge eating and inappropriate compensatory methods to prevent weight gain." [683] "The most common compensatory technique is the induction of vomiting after an episode of binge eating," but "[o]ther purging behaviors include the misuse of laxatives and diuretics." [684] A key component of the diagnosis is the patient's denial of the purging behavior, but with manifest physical signs or lab tests indicating the diagnosis. Some of these findings include dental erosion, palatal or oral trauma (from attempted induction of vomiting), abrasions along the backs of the hands (from attempted induction of vomiting), or electrolyte imbalances (from chronic vomiting or laxative use). [685] As with any other medical condition, there are other conditions on the differential diagnosis to consider prior to arriving at the diagnosis. These include anorexia nervosa, depression, gastrointestinal obstructive disorders, body dysmorphic disorder, Kluver Bucy syndrome, and gastrointestinal infectious *315 diseases. [686] It is the physician's task to consider these other disorders on the differential and order the appropriate labs and imaging prior to ruling them out [687] before arriving at the bulimia nervosa diagnosis. There is no question that bulimia nervosa is a valid diagnosis, or that a physician can reliably arrive at that diagnosis using the differential diagnosis methodology.

Much of what has been discussed above has been conceptual, esoteric, and possibly even vague. Perhaps an example of how the physician employs the differential diagnosis methodology in an AHT/SBS case will assist in clarifying the issue of its reliability. A three-month-old infant presents to the emergency room for "stopping breathing" (apnea). The mother's boyfriend, who was caring for the child while the mother was at work, states that the infant was crying. When he gave the infant a bottle, the infant "choked and gagged" and then "stopped breathing." He "shook" the infant gently to revive the infant. When the infant began crying a short time later, he soothed the infant and waited for the mother to return home, which occurred some hours later. When the mother returned home, the infant appeared pale and lethargic, and so the mother and her boyfriend proceeded to the ER for evaluation.

At the ER, the mother and her boyfriend denied any trauma for the infant in the prior three months of life. The mother denied any other problems in the child's medical history or any notable family medical history. On physical examination, the child was noted to have a small amount of swelling to the back of the head, but nothing else notable on physical examination--no bruising, scars, or other lesions. A head CT scan performed in the ER revealed an acute (fresh) subdural hemorrhage (SDH) along the front of both brain hemispheres and in between them (interhemispheric) and developing cerebral edema (brain swelling). The child was admitted for further *316 evaluation and management, and CPS was called. Further hospital evaluation, including whole body x-rays (a skeletal survey), revealed healing rib fractures on the right side of the rib cage. Ophthalmologic exam by the pediatric ophthalmologist revealed severe retinal hemorrhages (RHs) in both eyes. A child abuse pediatrician was consulted.

In this scenario, which is not different from many child abuse cases, the child abuse pediatrician is presented with multiple findings [688] --soft tissue swelling to the head, acute SDH, brain swelling (cerebral edema), severe RHs, and rib fractures--all of which have their own differential diagnoses. [689] For example, soft tissue swelling of the scalp has a limited differential diagnosis--trauma, infection (such as fungal or bacterial), inflammatory conditions, and dermatologic conditions (such as epidermal inclusion cysts). SDHs and RHs have a more expansive differential, [690] which can be generally characterized as trauma, bleeding disorders, malignancy, infection, and metabolic/genetic diseases. Rib fractures have a limited differential--trauma, genetic disease (such as osteogenesis imperfecta), nutritional deficiency (which results in weakened bone health and predisposes bones to fracture with mild trauma), prematurity, and medical procedures (such as cardiopulmonary resuscitation). [691] And cerebral edema does not itself have a differential diagnosis, but is rather a complex pathophysiological response to brain injury (resulting either primarily from direct trauma or as a *317 secondary response to lack of blood oxygen and blood). [692]

After the formulation of appropriate differential diagnoses for the relevant medical findings, it is at this point that the child abuse pediatrician engages in an inferential and deductive reasoning process that is in some aspects Bayesian and some aspects not. [693] In ruling out certain conditions on the differential, a physician may only utilize historical information, such as whether or not the child was born prematurely to rule out "prematurity," or whether or not CPR was performed to rule out "medical procedures" (as differential diagnoses for the rib fractures). In other cases, the physician may utilize historical information in combination with physical exam findings to rule out certain conditions. For example, in ruling out fungal or bacterial infection (as differential diagnoses for the soft-tissue swelling on the head), a physician would utilize the presence or absence of fever and the presence or absence of physical exam skin findings indicative of infection (like redness, warmth, bogginess, or the presence of blisters, vesicles, or scales). And, in other cases, the physician would combine historical information, physical exam findings, and laboratory information to rule out other conditions on the differential--such as using the absence of any historical symptoms, physical findings of lymph nodes or liver or spleen enlargement, and a normal white blood cell count (a lab) to rule out leukemia (on the differential for SDHs and RHs).

All these manners of eliminating certain conditions from the various differential diagnoses are non-Bayesian, i.e., there is no statistical quantification (pre and post-test probabilities) of the singular (e.g., the presence or absence of premature history in eliminating "prematurity") or cumulative (e.g., using historical, physical exam information, and the white blood count to eliminate "leukemia") probabilities of these diagnostic factors in the diagnostic process. Yet these are very reliable methods of eliminating some of the conditions from the differential diagnosis. They are rooted in years of experience with the known pathophysiological processes of disease and the *318 human body. And often, they are sanctified in medical treatises. [694] This is simply a reminder that the mere absence of linear, Bayesian analysis does not connote unreliability.

But there are aspects of the AHT/SBS differential diagnosis methodology that are conducive to Bayesian analysis. For example, in order to rule out bleeding disorders as a possible cause of SDHs in our scenario, it is informative to calculate the probability that a given bleeding disorder causes intracranial hemorrhage (ICH) in the general population. If we remember from the Bleeding

Disorders section above, this is attainable by multiplying the prevalence of that bleeding disorder by the prevalence of ICH in that bleeding disorder: [695]
(Prevalence of bleeding disorder) x

(Prevalence of ICH in that bleeding disorder)

For example, as noted in the Bleeding Disorders section above, the prevalence of Hemophilia A (Factor VIII) in the male population is 1 in 5000. [696] Also as noted in that section, the literature demonstrates that between 5-12% of these individuals will get an ICH at some time in their life. [697] Thus, assuming the highest prevalence (12%), the estimated probability that a person will get an ICH due to Hemophilia A is 2.4 per 100,000. But that is a lifetime risk assessment for hemophiliacs. If even 10% of these intracranial hemorrhages occurred in the first year of life, the rate would be only 24.1 in every 100,000 boys (or about 1 in 4 million). This is substantially lower than the incidence of SDHs attributable to AHT/SBS in the first year of life--which ranges from 24-29/100,000. [698] Comparatively, SDHs are about 100 times more likely to ***319** be secondary to AHT/SBS than Hemophilia A. And this is assuming the child has Hemophilia A. If lab testing rules out this diagnosis, then this comparative analysis is moot. Similar comparative ratios are available for all the bleeding disorders based upon the table listed in the Bleeding Disorders section above. [699]

Another example of Bayesian analysis is the probability of differentiating accidental trauma from AHT based upon the presence of the severe RHs. Here again, the evidence-based literature is instructive for physicians. Maguire et al. conducted a systematic review of RHs in AHT and accidental trauma. [700] The authors used strict inclusion criteria to identify 62 studies that represented 998 children aged 0-11 years, many of which were comparative studies between cohorts of accidental and non-accidental injury patients. [701] The authors only included studies where abuse was witnessed, admitted, or confirmed through multidisciplinary assessment and, in the comparative studies with accidents, where the accidents were witnessed. [702] This ensured minimization of 'circularity' in diagnosis by not relying on clinical features in the diagnostic assessment. [703] The authors performed multilevel logistic regression so that the data would be "more strongly correlated within the studies than between the studies." [704] Based upon their meta-analysis, they found that the probability of abuse in a child with head trauma and RHs was ninety-one percent. [705]

However, rather than computing statistics (the probability of ***320** abuse) that were limited to the population of patients in their study, the authors also calculated an odds ratio of abuse--a statistic that is generalizable to all populations of patients. [706] The authors determined that a child with head trauma and RHs has an odds ratio of 14.66 (95% CI: 6.39-33.62) for abuse. [707] In other words, if you know what the likelihood of abuse is for children admitted to your own Pediatric Intensive Care Unit (PICU), ER, or clinic with trauma, and you then find that one of those children has RHs, the likelihood that the child has been abused is now 14.6 times greater than that prior probability.

And these are not the only evidence-based statistics that physicians consider. Well-conducted systematic reviews discriminating inflicted from accidental injury have concluded that in a child with intracranial injury, apnea has an odds ratio of seventeen for abuse (with a positive predictive value of 93%) and rib fractures have an odds ratio of three for abuse. [708] While it may seem that these ratios (odds ratio of fourteen, seventeen, or three) are numerically not that high, they are actually rather impressive. For example, there is a proven benefit that psychosocial interventions (such as self-help material and telephone support) help people with ischemic heart disease quit smoking. [709] The evidence was so compelling for these strategies that they are now routine practice. Yet, the odds ratio for behavioral therapies was only 1.69 (95% CI 1.33 to 2.14), for telephone support 1.58 (95% CI 1.28 to 1.97), and for self-help only 1.48 (95% CI 1.11 to 1.96). [710] Taken in this context, the odds ratio for abuse with specific associated clinical features (apnea, retinal hemorrhages) is extremely compelling.

Finally, and most importantly, evidence-based literature has quantitatively specified that which common sense has qualitatively *321 known: In a child with traumatic intracranial injury, the absence of a trauma history has a 97% specificity and 92% positive predictive value for abuse.[711] A predictive value is another statistic that is immensely helpful. It is the probability of a disease after additional information (such as from a test) has been obtained.[712] Thus, a positive predictive value is the probability of disease in those known to have a positive test result. Or in the above scenario, in the child with traumatic intracranial injury, the absence of trauma history (a.k.a., the positive test) indicates a 92% probability of abuse.

While it may seem that the statistics are dispositive, they are actually only a portion of the analysis. Physicians must remain mindful of "conjunction fallacies," "overweighting," "errors in [the] revision of probabilities," and other pitfalls.[713] Cognizance of these potential errors minimizes the probability of their occurrence.

Ultimately, the differential diagnosis methodology is a marriage of evidence-based literature and experience; a symbiosis of inferential and deductive reasoning; a synergy of linear and non-linear dynamic thought. It is the methodology by which physicians achieve diagnostic sufficiency.[714] So with the information presented, the questions for the reader and the gatekeeper are: Is the methodology presented "junk science?" Is it not even "good enough to be wrong?"

The reliability of the differential diagnosis methodology, a methodology utilized by all physicians--not just child abuse pediatricians, has been laid out for the reader to judge for himself or herself. For countless physicians, this methodology is reliable. In general, the application of this methodology to the facts of a case *322 comprises the "minor premise" of the AHT syllogistic argument. The sound application of this methodology to a particular set of facts is for the individual, particular the gatekeeper, to determine.

C. The "Path Forward": Throwing the Baby out with the Bath Water (Figuratively Speaking)

Findley et al. have suggested that our disagreement on this issue (AHT/SBS) is "narrow but critical."[715] We could not disagree more. Our disagreement lies not just in a misunderstanding of the quality or sufficiency of the medical literature; it represents a vast philosophical and ideological difference about the value and roles of clinical judgment and our current jury system in cases involving medical expert testimony. We shall examine the logical shortcomings of Findley et al.'s positions and discuss our recommendations for resolving Daubert issues in AHT/SBS cases.

1. The fog of legal argument

There is an old defense adage: "If you can't win on the facts, argue the law; if you can't win on the law, then just confuse everyone." The applicability of this axiom will hopefully be apparent.

Findley et al. bemoan the "subjective" and unreliable nature of clinical judgment.[716] The authors state that its "subjective nature" is not "the objective medical evidence envisioned by evidence-based medicine and Daubert" and would ultimately "result in mistaken diagnoses and false convictions."[717] Despite making this assertion repeatedly,[718] the authors then conclude that certain clinical judgments (in AHT/SBS cases) are "obvious,"[719] without defining by what criteria they become "obvious," if clinical judgment is "obviously" unreliable. Additionally, if "subjectivity" spells the death of clinical *323 judgment, then what physician testimony could ever survive? Such reasoning would necessitate the undesirable exclusion of all physician testimony--the pediatrician, the emergency room physician, the neurosurgeon, the ophthalmologist, the radiologist, and the forensic pathologist.

Findley et al. invoke this same fallacy--hasty generalization--in their argument for the invalidation of the AHT/SBS diagnosis. The authors spend considerable effort arguing that biomechanics has conclusively demonstrated that shaking does not even

come close to reaching the thresholds for causing concussive injury or SDHs.[720] Then Findley et al. generalize this premise--that shaking cannot cause SDHs--as the basis for invalidating the entire AHT/SBS diagnosis.[721] Yet, despite proffering the diagnosis as invalid, as mentioned above, the authors nevertheless concede that some AHT/SBS cases are "obvious."[722] This begs the question of how some cases are "obvious" if the diagnosis is in fact invalid.

Interestingly, the confusion takes a different turn. Whereas Findley et al. are adamant that science has demonstrated that shaking is not dangerous, the authors then ultimately concede that "violent shaking" is "dangerous," and that "there are few costs and many potential benefits associated with educating parents that they should never shake a child."[723] With that inconsistency being apparent, questions arise: If shaking does not even come close to concussive or SDH-causative forces, then what exactly do Findley et al. believe shaking is "dangerous" enough to cause? A bruise? A neck sprain? If shaking is not dangerous enough to cause serious injury, then why are prevention programs necessary or recommended? To prevent a potential bruise or neck sprain? Considerable medical research resources have already been spent to validate something that res ipsa loquitur sufficiently explains--that shaking a young infant can and does cause serious intracranial injury. It is careful, deliberate, and ***324** reasoned medical judgment through the differential diagnosis methodology that helps to eliminate potential alternative causes for those injuries.

2. Lifting the fog

Married with this confusion is a healthy distrust of the jury system. Findley et al. state:

> This approach [experts with differing perspectives arguing it out in the courtroom] presents two problems. First, trying and retrying undecided scientific issues on a weekly basis is extraordinarily expensive and inevitably results in inconsistent and 'fluky' justice. Second, and perhaps more important, if doctors cannot agree on these complex and unresolved issues, it is unlikely that jurors or judges can do any better.[724]

Findley et al. argue that the resolution to this issue, or "the path forward" or "getting it right," is to "acknowledge the complexities" and, when applicable, to say "we don't know."[725] Setting aside the veiled professional adhominem that physicians do not already do this (and consequently, are diagnosing and testifying carelessly, capriciously, maliciously, or at best, erroneously), the logical consequence of this course is diagnostic and testimonial abstinence. But is that really "getting it right?" Or is that just a safer course for those who are alleged or actual perpetrators of abuse? Do diagnostic and testimonial abstinence not then end up supplanting one "fluky justice" for another? It seems to us that justice should balance the "actual innocents" on both sides of the scale, not just those potentially accused of child abuse.

Thus, before we "toss the baby out with the bathwater," we propose improvements in our current constructs, both medical and legal, as a true path forward. From the medical construct, first and foremost, there must be increased funding for child maltreatment research at both the state and federal levels.[726] Increased funding for ***325** evidence-based research in the forensic disciplines was a primary recommendation by the National Research Council.[727] The extension to the field of child maltreatment is a natural corollary. At present, the Institute of Medicine is already investigating avenues of needed research in the field of child maltreatment.[728] However, it cannot be emphasized enough that the need for further research is not an indictment of the quality of the current research. As demonstrated above, multiple research collaboratives have enhanced the current evidence-based literature such that we are on the verge of clinical decision rules in AHT/SBS.[729]

Second, as Narang mentioned in his first article[730] (and Findley et al. have echoed),[731] a multidisciplinary body, under the auspices of the National Academy of Sciences and/or the National Institute of Health, needs to make a global assessment of the evidence-based literature on AHT/SBS and promulgate its findings. Third, medical professional societies must take a more active role in the regulation of irresponsible testimony by its members. One organization, the American Association of

Neurologic Surgeons, has been a model for the societies in imposing disciplinary actions against its members for irresponsible expert testimony. [732] Other societies need to follow suit. Finally, physicians need to engage in reciprocal intra-disciplinary and inter-disciplinary educational efforts on multiple medico-legal topics in AHT/SBS, to include but not limited to, implicit bias in medical decision making, evidence-based literature and AHT/SBS, and responsible expert testimony in AHT/SBS.

With regard to the legal construct, making inroads into the problem of ensuring reliable medical expert testimony requires travel *326 along several paths. The Supreme Court and legal scholars have clearly recognized that Daubert has not turned out to be the panacea it was believed it would be. Thus, with the recent ruling in Melendez-Diaz, [733] some scholars assert the Court has sought to remedy Daubert's shortcomings by strengthening the confrontation rights of the accused. [734] While emboldening the Confrontation Clause is certainly an acceptable adjunct, emboldening the gatekeeper and the jury are valuable objectives as well. One important avenue for achieving those objectives is the increased utilization of FRE 706. The assistance of the independent expert to the court (for Daubert purposes) and to the jury (for any questions that may arise in the course of the adversarial process) could well serve the goals of truth and justice. This sage counsel was offered by Judge Learned Hand over a hundred years ago, [735] but this counsel has found spotty adherence at best.

As with the medical construct, another important consideration is the need for more legal/social science research. Bold assertions have been made of a judicial system that is "riddled with false convictions." [736] But in order to adequately assess the validity of such statements, or at least to adequately grasp the scope of the problem, a denominator is needed. Certainly, false convictions have occurred. But in what percentage of cases? Currently, there is only conjecture. Not only is that research needed, but further research is needed into why these false convictions have occurred, what variables are most common in those cases, and what systemic changes would be effective in minimizing those cases. Finally, also as with medicine, additional education for the gatekeeper (on topics such as the basics of good scientific studies, ongoing advances in the science related to AHT/SBS, etc.) would be beneficial.

*327 VIII. Conclusion

What has been presented for the reader in Narang's first article and in this article is a reasonable summary of the evidence-based literature that girds the AHT/SBS diagnosis, an analysis of the strengths and limitations of that literature, and a detailed Daubert analysis of the differential diagnosis methodology by which physicians arrive at that diagnosis. These comprise the major and minor premises of the syllogistic structure of the expert's opinion offered in court. For countless physicians and professional medical societies, they are valid and reliable. What has also been presented, in contrast, is the lack of evidence-based literature for alternative hypotheses--such as hypoxia, the immature dural vascular plexus theory, dysphagic choking, and "neck" injury.

AHT/SBS cases are complex, difficult cases. Physicians engaged in such cases do "acknowledge the complexities" and "consider alternative medical causes." It is exactly those precepts that delayed the medical and societal recognition of abusive injury as the correct diagnosis in the early historical cases. [737] Now, some scholars argue for a return to those times as a "path forward." But diagnostic and testimonial abstinence has had its day on the scientific stage. It has had its day on the differential . . . and science has ruled it out.

Footnotes

1 The authors' use of the terminology "Abusive Head Trauma/Shaken Baby Syndrome" is not to imply that the terms are interchangeable. It is simply to recognize that there is a commonly recognized subset of Abusive Head Trauma-- Shaken Baby Syndrome--that is the primary subject of controversy, and that pediatricians have transitioned to a more encompassing term--Abusive Head Trauma. See, e.g., Emily Bazelon, Shaken Baby Syndrome Faces New Questions in Court, N.Y. Times, Feb. 2, 2011, available at http:// www.nytimes.com/2011/02/06/magazine/06baby-t.html?_r=1.

2 Id.; Deborah Tuerkheimer, Anatomy of a Misdiagnosis, N.Y. Times, Sept. 20, 2010, available at www.nytimes.com/2010/09/21/opinion/21tuerkheimer.html?ref=opinion.

3 See generally Keith A. Findley et al., Shaken Baby Syndrome, Abusive Head Trauma, and Actual Innocence: Getting It Right, 12 Hous. J. Health L. & Pol'y 209 (2012); Deborah Tuerkheimer, The Next Innocence Project: Shaken Baby Syndrome and the Criminal Courts, 87 Wash. U. L. Rev. 1 (2009).

4 See, e.g., State v. Edmunds, 746 N.W.2d 590 (Wis. Ct. App. 2008) (finding that a "significant and legitimate debate in the medical community has developed in the past ten years"); Hamilton v. Commonwealth, 293 S.W.3d 413 (Ky. Ct. App. 2009) (holding error to permit testimony on shaken baby syndrome without first conducting a Daubert hearing since no Kentucky case had specifically determined it was a "reliable" theory).

5 Cavazos v. Smith, 132 S.Ct. 2, 10 (2011).

6 Id.

7 J. Moreno & B. Holmgren, Dissent into Confusion: The Supreme Court, Pseudoscience, and the False Shaken Baby Syndrome Controversy, Utah L. Rev. (forthcoming 2013).

8 Stephen Breyer, Introduction, in Reference Manual on Scientific Evidence, Second Edition 1-8, 4 (2000) (citation refers only to the quoted words "[not] even good enough to be wrong").

9 Moreno & Holmgren, supra note 7. (providing a detailed analysis of the rationales and shortcomings of the dissenting justices' opinion in Cavazos v. Smith).

10 Sandeep Narang, A Daubert Analysis of Abusive Head Trauma/Shaken Baby Syndrome, 11 Hous. J. Health L. & Pol'y 505, 505 (2012).

11 Id. at 578.

12 Id. at 541-48.

13 Id. at 548-58.

14 Id. at 574-76.

15 Id. at 576-83.

16 Id. at 628-29.

17 See generally L. Frasier et al., Abusive Head Trauma In Infants & Children: A Medical, Legal, and Forensic Reference (G. W. Medical Pub. 2006); L. Rorke-Adams et al., Head Trauma, in Child Abuse: Medical Diagnosis & Management 53-119 (R. Reece & C. Christian eds., American Academy of Pediatrics 2009); C. Jenny, Child Abuse and Neglect: Diagnosis, Treatment, and Evidence, ch. 6, 39-48 (2010).

18 See Oxford Centre for Evidence-based Medicine, Levels of Evidence (March 2009), www.cebm.net/index.aspx?o=1025 (last updated Jan. 29, 2013) (last visited March 1, 2013).

19 "Hypoxia" is defined as low tissue oxygenation. See Christian Rosenberger et al., Immunohistochemical Detection of Hypoxia-Inducible Factor-1a in Human Renal Allograft Biopsies, 18 J. Am. Soc. Nephrol. 343, 349 (2006); "Ischemia" is defined as a deprivation of blood supply to body tissue. See, e.g., David J. Hearse, Ischemia, Reperfusion, and the Determinants of Tissue Injury, 4 Cardiovascular Drugs & Therapy 767, 768 (1990).

20 Hippocrates, Of the Epidemics, (University of Adelaide Library 2007), available at http://ebooks.adelaide.edu.au/h/hippocrates/edpidemics/ (last updated Sept. 16, 2012; last visited March 1, 2013).

21 See, e.g., id.

22 The Lancet, a prominent medical journal, was first published October 5, 1823. See About the Lancet Medical Journal, Elsevier, Inc., http:// www.thelancet.com/lancet-about.

23 David L. Sackett et al., Evidence Based Medicine: What It Is and What It Isn't, 312 BMJ 71, 71 (1996) (emphasis added).

24 See, e.g., Brad Petrisor & Mohit Bhandari, The Hierarchy of Evidence: Levels and Grades of Recommendation, 41 Indian J. Orthopaedics 11, 11 (2007).

25 See Sackett et al., supra note 23, at 72.

26 See Petrisor & Bhandari, supra note 24, at 11-12.

27 See, e.g., David Moher et al., Preferred Reporting Items for Systematic Reviews and Meta-Analysis: The PRISMA Statement, 6 PLoS Med. 1, 4-5 (2009), available at http://www.plosmedicine.org/article/info%3Adoi%C2F10.1371% 2Fjournal.pmed.1000097#s4 (systemic reviews and meta analysis); Kenneth F. Schulz, CONSORT 2010 Statement: Updated Guidelines for Reporting Parallel Group Randomized Trials, 152 Annals of Internal Med. 726, 727 (2010), available at http://annals.org/article.aspx? articleid=746833; Erik von Elm et al., The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) Statement: Guidelines for Reporting Observational Studies, 61 J. Clinical Epidemiology 344, 346-47 (2008), available at http:// download.journals.elsevierhealth.com/pdfs/journals/0895-4356/PIIS0895435607004362.pdf (observational studies including cohort, case-control, and cross-sectional studies).

28 This was the type of scale utilized by Dr. Donohoe in Mark Donohoe. Evidence-Based Medicine and Shaken Baby Syndrome Part 1: Literature Review, 24 Am. J. of Med. Pathology 239, 240-41 (2003), and was heavily relied upon by the dissenting justices in Cavazos v. Smith, as well as by other legal scholars. See Cavazos, 132 S. Ct. 2,10 (2011); Tuerkheimer, supra note 3, at 12-13; See also Molly Gena, Shaken Baby Syndrome: Medical Uncertainty Casts Doubt on Convictions, 3 Wis. L. Rev. 701, 706, 710, 727 (2007).

29 See Oxford Centre for Evidence-Based Medicine, supra note 18.

30 Id.

31 Id.

32 Bob Phillips et al., Levels of Evidence (March 2009), Oxford Centre for Evidence Based Medicine (Aug. 2, 2013), http:// www.cebm.net/index.aspx? o=1025.

33 See, e.g., Donohoe, supra note 28.

34 See Donohoe, supra note 28, at 240.

35 Bob Phillips et al., supra 32.

36 Mark B. McClellan et al., Evidence-Based Medicine and the Changing Nature of Healthcare, 2007 IOM Annual Meeting Summary, available at http:// www.nap.edu/catalog/12041.html.

37 See Findley et al., supra note 3, at 305-06.

38 Public health advocates, for various reasons, prefer the term "preventable injury." For the purposes of this paper, and with no intent to diminish the laudable public health reasons for the use of that terminology, we shall use the term "accident," as it is the more commonly utilized term in the medical literature in comparison to abusive injury.

39 See David L. Chadwick et al., Annual Risk of Death Resulting From Short Falls Among Young Children: Less than 1 in 1 Million, 122 Pediatrics 1213 (2008).

40 Takeo Fujiwara et al., Characteristics That Distinguish Abusive From Nonabusive Head Trauma Among Young Children Who Underwent Head Computed Tomography in Japan, 122 Pediatrics 841, 842-43 (2008).

41 Much as all cancers are not the same, all "short falls" are not the same. Stairway falls, falls from moving objects (such as shopping carts or moving strollers or walkers), or falls involving occipital (back of the head) impact do not entail the same biomechanics as

simple short falls off furniture (such as beds or couches), where typically the frontal or parietal (side) areas of the skull are impacted. The varying biomechanical forces on different anatomic structures (such as the head, the neck, or the torso) in stairway falls, falls from moving objects, or falls with occipital impact warrant their distinction into a separate category than the "simple short fall." Hereinafter, unless otherwise stated, the literature reviewed pertains to simple short falls.

42 Michael Y. Wang et al., Injuries From Falls in the Pediatric Population: An Analysis of 729 Cases, 36 J. Pediatric Surgery 1528, 1528-29 (2001).

43 John Plunkett, Fatal Pediatric Head Injuries Caused by Short Distance Falls, 22 Am. J. Forensic Med. Pathology 1, 10 (2001); Gregory Reiber, Fatal Falls in Childhood: How Far Must Children Fall to Sustain Fatal Head Injury? Report of Cases and Review of the Literature, 14 Am. J. Forensic Med. Pathology 201, 201 (1993).

44 Chadwick et al., supra note 39 at 1213.

45 See id. at 1213.

46 See, e.g., id.

47 See, e.g., id.; see also S.A. Warrington et al., Accidents and Resulting Injuries in Premobile Infants: Data From the ALSPAC Study, Archives of Disease in Childhood 104, 104 (2001); Julia Wrigley & Joanna Dreby, Fatalities and the Organization of Child Care in the United States, 70 Am. Sociology Rev. 729, 743-49 (2005).

48 See, e.g., id. at 1220. Some of these methodological variances have included variations in short fall definition, variations in inclusion and exclusion criteria of patients, and variations in outcome aspects.

49 This section is not intended to be an exhaustive review of the topic as it is outside the scope and purpose of this paper. For a more thorough review of the topic, see Child Abuse and Neglect: Diagnosis, Treatment, and Evidence 39-48 (C. Jenny et al., eds., 2010); Abusive Head Trauma in Infants and Children: A Medical, Legal, and Forensic Reference (L. Frasier et al. eds., 2006); Child Abuse Medical Diagnosis and Management 53-119 (Robert M. Reece et al. eds., 3d ed. 2009) .

50 Hereinafter, the use of the terms "severe injury" or "serious injury" refer to their meaning within the AIS (Abbreviated Injury Scale). See Abbreviated Injury Scale, Association for the Advancement of Automotive Medicine (Oct. 28, 2012), http://www.aaam1.org/ais/; Abbreviated Injury Scale, TRAUMA.ORG (Oct. 28, 2012), http://www.trauma.org/archive/scores/ais.html. Although it is common, clinically, to incorporate the AIS score into an Injury Severity Score (ISS) when assessing overall trauma to the human body, for the purposes of this article, "severe injury" or "serious injury" will refer to their use within the AIS. Although the AIS dictionary has specific codes for specific head injuries, typically intracranial hemorrhages have scores of either 3 or 4 (3 = serious; 4 = severe) and cerebral edema (brain swelling) has a score of 5 (critical). See Thomas Songer, Measuring Injury Severity (Oct. 28, 2012), available at http://www.pitt.edu/~epi2670/severity/severity.pdf.

51 Suzanne B. Haney et al., Characteristics of Falls and Risk of Injury in Children Younger Than 2 Years, 26 Pediatric Emergency Care 914, 914-15 (2010).

52 Id. at 915.

53 Id. at 917.

54 Id. at 918.

55 S.A. Warrington et al., supra note 47, at 104.

56 Id. at 105.

57 Id. at 104.

58 Id. at 105.

59 Id.

60 Id. at 106-07.

61 See Harvey Kravitz et al., Accidental Falls from Elevated Surfaces in Infants from Birth to One Year of Age, 44 Pediatrics 869 (1969); Ray E. Helfer et al., Injuries Resulting from when Small Children Fall Out of Bed, 60 Pediatrics 533 (1977).

62 See Oxford, supra note 18.

63 Certainly, a limitation of the study was that the data was subject to recall inaccuracy or bias. But, there was little concern for intentionally skewed data (as is a concern in suspected AHT cases).

64 Thomas J. Lyons & R. Kim Oates, Falling Out of Bed: A Relatively Benign Occurrence, 92 Pediatrics 125, 126 (1993).

65 Id. at 126.

66 Id.

67 Id.

68 Prasit Nimityongskul & Lewis Anderson, The Likelihood of Injuries When Children Fall Out of Bed, 7 J. Pediatric Orthopedics 184, 184-86 (1987).

69 S. Levene & G. Bonfield, Accidents on Hospital Wards, 66 Archives of Disease in Childhood 1047, 1047-1049 (1991).

70 S. Monson et al., In-Hospital Falls of Newborn Infants: Data From a Multihospital Healthcare System, 122 Pediatrics 227, 278-280 (2008).

71 C. Ruddick et al., Head Trauma Outcomes of Verifiable Falls in Newborn Babies, 95 Archives of Disease in Childhood Fetal Neonatal Ed. 144, 144-45 (2010).

72 P.L. Schaffer et al., Pediatric Inpatient Falls and Injuries: A Descriptive Analysis of Risk Factors, 17 J. Special Pediatric Nursing 10 (2012).

73 One author has asserted that hospital settings may not be "objective," as there is an equal likelihood for malicious acts by nursing or other staff in hospitals as in any other setting. See J. Ehsani et al., The Role of Epidemiology in Determining if a Simple Short Fall Can Cause Fatal Head Injury in an Infant, 31 Am. J. Forensic Pathology 287, 290 (2010). However, hospital settings are by nature, less private settings, with clear understanding to all within those settings of a diminished level of privacy. Given that abusive acts occur in private settings, it is unreasonable to assume an equal likelihood of abuse in inherently less private/more public settings.

74 Julia Wrigley & Joanna Dreby, Fatalities and the Organization of Child Care in the United States, 70 Am. Sociology Rev. 729, 743-49 (2005).

75 See Chadwick, supra note 39.

76 Id.

77 Id. at 1216.

78 Good et al.'s review found nine deaths in child care centers, but six were attributed to natural causes and three to "unintentional" causes (two asphyxiation and one pedestrian/MVA). See Susan E. Good et al., Children's Deaths at Day-care Facilities, 9 Pediatrics, 1039, 1039-40 (1994).

79 Wrigley & Dreby, supra note 74, at 749.

80 See Chadwick, supra note 39, at 1216.

81 U.S. Department of Education, Child Care and Early Education Arrangements in Infants, Toddlers, and Preschoolers: 2001 (2001), available online at http://nces.ed.gov/pubs2006/2006039.pdf (last visited Sept. 5, 2012).

82 See Chadwick, supra note 39.

83 R. A. Williams, Injuries in Infants and Small Children Resulting from Witnessed and Corroborated Free Falls, 31(1) J. Trauma 1350, 1350 (1991).

84 Id. at 1351.

85 Id.

86 Id.

87 Id.

88 See Wrigley & Dreby, supra note 74.

89 Robert M. Reece & Robert Sege, Childhood Head Injuries: Accidental or Inflicted?, 154 Archive Pediatric Adolescent Med. 11, 11 (2000).

90 K. Johnson et al., Accidental Head Injuries in Children Under 5 Years of Age, 60 Clinical Radiology 464, 464 (2005).

91 See Williams, supra note 83, at 1351.

92 David L. Chadwick et al., Deaths from Falls in Children: How Far is Fatal?, 31 J. Trauma 1353, 1353-55 (1991).

93 Id. at 1354. An 11-month-old child was found dead beneath a second story window. Parents were not aware of the child's absence until notified by neighbors who found the child.

94 Id.

95 Id.

96 See Reece & Sege, supra note 89, at 11.

97 See Nicole G. Ibrahim et al., Influence of Age and Fall Type on Head Injuries in Infants and Toddlers, 30 Int'l J. Dev. Neuroscience 201, 201-206 (2012) (proposing that the fall type and age of child are important factors in the resulting injury of a child); see also James A. Murray et al., Pediatric Falls: Is Height a Predictor of Injury and Outcome?, 66 Am. Surgeon 863, 863-865 (2000).

98 See Angela K. Thompson et al., Pediatric Short-Distance Household Falls: Biomechanics and Associated Injury Severity, 43 Accident Analysis Prevention 143, 143 (2011); see also Johnson et al., supra note 90, at 467; Michael Y. Wang et al., Injuries from Falls in the Pediatric Population: An Analysis of 729 Cases, 36 J. Pediatric Surgery 1528, 1528 (2000).

99 A.C. Duhaime et al., Head Injury in Very Young Children: Mechanisms, Injury Types, and Ophthalmologic Findings in 100 Hospitalized Patients Younger than 2 Years of Age, 90 Pediatrics 179, 179 (1992).

100 Id. at 180.

101 Id.

102 Id.

103 Id. at 181.

104 Id.

105 Id.

106 Id.

107 Id. Three of the twenty-six short falls had epidural hemorrhages (EDHs). Id. Of the twenty-one longer falls, six had very focal subarachnoid hemorrhages or brain contusions. Id. All fall children had benign hospital courses. Id.

108 Id. at 182.

109 Id.

110 Kirsten Bechtel et al., Characteristics that Distinguish Accidental from Abusive Injury in Hospitalized Young Children with Head Trauma, 114 Pediatrics 165, 165 (2004).

111 See Thompson et al., supra note 98, at 144.

112 Id. at 143.

113 Id. at 144.

114 Id.

115 Id. at 145. These were described as "lacerations" or "contusions."

116 Id. These were described as "fractures."

117 Id. These were described as "small isolated SDHs."

118 Id. at 143.

119 Id. at 149.

120 Id.

121 See Chadwick, supra note 39, at 1213.

122 Id.

123 Id. at 1215-19.

124 Id. at 1214. ("EPIC" stands for Epidemiology and Prevention for Injury Control Branch.)

125 Id. ("WISQRS" stands for Web-based Injury Statistics Query and Reporting System.)

126 Id.

127 See Chadwick, supra note 39, at 1214.

128 Id.

129 Id.

130 Id. at 1213.

131 From Short Falls Among Young Children: Less than 1 in 1 Million, 121 Pediatrics 1213, 1220 (2008). Reprinted with permission.

132 Johnathon P. Ehsani et al., The Role of Epidemiology in Determining if a Simple Short Fall can Cause Fatal Head Injury in an Infant, 31 Am. J. Forensic Med. Pathology 287, 287 (2010).

133 Id.

134 Id.

135 Id.

136 Id. The single case alluded to by the authors is a reported videotaped short fall resulting in death after a brief lucid interval in Plunkett's review of the U.S. Consumer Product Safety Commission database. See Plunkett, supra note 43, at 4; Ehsani, supra note 131, at 290.

137 See Plunkett, supra note 43, at 1.

138 Id. at 2.

139 Id. Since the database is geared towards detection of fatalities related to products, it can miss fatalities not related to products, and is not designed to discriminate false histories related to products (probably including abusive injuries within the data subset).

140 See Chadwick, supra note 39, at 1215. "Nine of the 18 children who died were [older than] 5 years of age. Among the 9 young children, 4 cases were not witnessed at all, even by other children. Of the remaining 5 cases, 1 fall height was estimated at [greater than] 2.0 m. Of the remaining 4 cases, 1 had no autopsy, and the cause of death in that case was uncertain.... With the determination that 3 of the cases were valid, the annual population risk for a short-fall death of a young child in this (playground) sample can be calculated as 3 fatalities/(400 000 x 12) = 0.625 cases per 1 million young children per year." Id.

141 Matthieu Vinchon et al., Confessed Abuse Versus Witnessed Accidents in Infants: Comparison of Clinical, Radiological, and Ophthalmological Data in Corroborated Cases, 26 Childs Nervous Sys. 637, 642 (2010).

142 See Matthieu Vinchon et al., Accidental and Nonaccidental Head Injuries in Infants: A Prospective Study, 102 J. Neurosurgery 380, 380 (Supp. 2005); Bechtel, supra note 110, at 165.

143 Kent P. Hymel et al., Head Injury Depth as an Indicator of Causes and Mechanisms, 125 Pediatrics 712, 713 (Supp. 2010).

144 Id.

145 Id. at 712-13.

146 Id. at 712. The odds ratio is quite notable in this study. It indicates that the finding of subcortical brain injury is thirty five times more likely to be the result of abuse than accident. The authors also found that subcortical injury (i.e. deeper brain injury) more frequently demonstrated inertial injury and manifested acute respiratory or circulatory compromise. Id. These findings had high statistical significance. Id.

147 See Kent P. Hymel et al., Derivation of a Clinical Prediction Rule for Pediatric Abusive Head Trauma, 14 Pediatric Critical Care Med. 210 (2013).

148 S. Maguire et al., What Clinical Features Distinguish Inflicted from Non-Inflicted Brain Injury? A Systematic Review, 94 Archives Disease Childhood 860, 860 (2009).

149 Id.

150 Id. at 861.

151 Id. at 861, 864.

152 Id. at 864.

153 Id. at 860.

154 Maguire, supra note 147, at 866. "Apnoea" refers to the cessation of breathing (usually defined as longer than twenty seconds in infants). Melissa Scollan-Koliopoulos & John S. Koliopoulos, Evaluation and Management of Apparent Life-Threatening Events in Infants, 36 Pediatric Nursing 77, 77 (March-April 2010).

155 Maguire, supra note 147, at 865.

156 Id. (emphasis added).

157 Id.

158 See generally Scott Denton & Darinka Mileusnic, Delayed Sudden Death in an Infant Following an Accidental Fall: A Case Report with Review of the Literature, 24 Am. J. Forensic Med. & Pathology 371 (2003); Horace B. Gardner, A Witnessed Short Fall Mimicking Presumed Shaken Baby Syndrome (Inflicted Childhood Neurotrauma), 43 Pediatric Neurosurg 433-35 (accepted after revision May 17, 2007);; Patrick E. Lantz & Daniel E. Couture, Fatal Acute Intracranial Injury, Subdural Hematoma, and Retinal

Hemorrhages Caused by Stairway Fall, 56 J. Forensic Sci. 1648 (2011); Patrick D. Barnes et al., Traumatic Spinal Cord Injury: Accidental Versus Nonaccidental Injury, 15 Seminars in Pediatric Neurology 178 (2008).

159 See generally Jan E. Leestma, Case Analysis of Brain-Injured Admittedly Shaken Infants: 54 Cases, 1969-2001, 26 Am. J. Forensic Med. & Pathology 199-211 (2005).

160 Sabine A. Maguire et al., Estimating the Probability of Abusive Head Trauma: A Pooled Analysis, 158 J. Pediatrics 550, 550 (2011).

161 Id. at 550.

162 C.A.B. Clemetson, Elevated Blood Histamine Caused by Vaccinations and Vitamin C Deficiency May Mimic the Shaken Baby Syndrome, 62 Med. Hypothesis 533, 534 (2004); Horace B. Gardner, Immunizations, Retinal and Subdural Hemorrhages: Are They Related?, 64 Med. Hypothesis 663, 663 (2005).

163 Michael O. Gayle et al., Retinal Hemorrhage in the Young Child: A Review of Etiology, Predisposed Conditions, and Clinical Implications, 13 J. of Emergency Med. 233, 237 (1995); Lois K. Lee et al., Intracranial Hemorrhage After Blunt Head Trauma in Children with Bleeding Disorders, 158 J. Pediatrics 1003, 1003 (2011).

164 See Carpenter et al., Technical Report: Evaluating for Suspected Child Abuse: Conditions that Predispose to Bleeding, 131 Pediatrics 1357 (2013), available at http:// pediatrics.aappublications.org/content/131/4/e1357.full.html.

165 Id. at 1368-69.

166 S. S. Acharya et al., Rare Bleeding Disorder Registry: Deficiencies of Factors II, V, VII, X, XII, Fibrinogen and Dysfibrinogenemias, 2 J. of Thrombosis and Haemostasis 248, 249 (2004).

167 Id. at 254-55.

168 See Monagle & Andrew, infra note 171.

169 Acharya, supra note 165, at 249.

170 See Carpenter et al., supra note 163, at 1357.

171 For a more detailed review of congenital bleeding disorders, see id.

172 D. B. Wilson, Acquired Platelet Defects, in Nathan And Oski's Hematology Of Infancy And Childhood 1557 (Stuart H. Orkin et al. eds., 6th ed., 2003); P. Monagle & M. Andrew, Acquired Disorders of Hemostasis, in Nathan And Oski's Hematology Of Infancy And Childhood 1631 (Stuart H. Orkin et al. eds., 6th ed., 2003).

173 Immune Thrombocytopenic Purpura (ITP) is a condition in which the body forms antibodies to the blood platelets, resulting in their destruction.

174 Disseminated Intravascular Coagulation (DIC) results from disordered clotting and bleeding, and this can be secondary to a variety of reasons--overwhelming infection, severe trauma, anaphylaxis, etc. It occurs only in children who are severely ill, and may result in bleeding in any part of the body, including intracranial bleeding. Monagle & Andrew, supra note 171, at 1633-35.

175 Vitamin K Deficiency bleeding (previously known as Hemorrhagic Disease of the Newborn) is a bleeding disorder that occurs most commonly in the newborn/early infancy period. Newborns are born with low levels of vitamin K, an essential factor in blood clotting. Unless provided with intramuscular dose of Vitamin K in the newborn period, young infants can suffer bleeding from mucosal surfaces, bleeding from circumcision, generalized bruising, large intramuscular hemorrhages, and intracranial hemorrhage. Id. at 1635-37.

176 "Coagulopathy" can best be defined as a disorder of the natural hemostasis of the body. Since the liver produces some of the clotting factors needed to clot blood, severe liver dysfunction can result in a "coagulopathy."

177 Amy A. Hassan & Michael H. Kroll, Acquired Disorders of Platelet Function, Am. Soc'y of Hematology of Educ. Program Book 403, 404-05 (2005).

178 Id. at 403.

179 See Monagle & Andrew, supra note 171, at 1638.

180 Id.

181 Martin J. Shearer, Vitamin K Deficiency Bleeding (VKDB) in Early Infancy, 23 Blood Rev. 49, 50 (2009).

182 Id. at 53. In patients who have already received vitamin K as treatment or transfusion of plasma, measurement of proteins induced by vitamin K absence (PIVKA II) can confirm the diagnosis.

183 Id. at 50-51.

184 Cindy Neunert et al., The American Society of Hematology 2011 Evidence-Based Practice Guidelines for Immune Thrombocytopenia, 117 Blood 4190, 4195 (2011).

185 Alvin Zipursky, Prevention of Vitamin K Deficiency Bleeding in Newborns, 104 Brit. J. Haematology 430, 432-33 (1999).

186 Heather McKay et al., Bleeding Risks Associated with Inheritance of the Quebec Platelet Disorder, 104 Blood 159, 164 (2004).

187 Id. at 164.

188 Pravas Mishra et al., Intracranial Haemorrhage in Patients with Congenital Haemostatic Defects, 14 Haemophilia 952, 952-53 (2008); Marvin D. Nelson Jr. et al., Prevalence and Incidence of Intracranial Haemorrhage in a Population of Children with Haemophilia, 5 Haemophlia 306, 311 (1999); Rachelle Nuss et al., Changes in the Occurrence of and Risk Factors for Hemophilia-Associated Intracranial Hemorrhage, 68 Am. J. Hematology 37, 40 (2001).

189 See Carpenter et al., supra note 163, at 1358-1362.

190 Roshni Kulkarni et al., Sites of Initial Bleeding Episodes, Mode of Delivery and Age of Diagnosis in Babies with Haemophilia Diagnosed Before the Age of 2 Years: A Report from the Centers for Disease Control and Prevention's (CDC) Universal Data Collection (UDC) project, 15 Haemophilia 1281, 1281 (2009).

191 A.D. Metjian et al., Bleeding Symptoms and Laboratory Correlation in Patients with Severe von Willebrand Disease, 15 Haemophilia 918, 919 (2009).

192 Ideally, the scientific literature would include studies that have systematically compared trauma histories, locations of bleeding, amount of bleeding, clinical presentations and outcomes, and the presence of retinal hemorrhages in children with and without bleeding disorders. However, given the rarity of most bleeding disorders, such studies would be extremely difficult and costly to conduct; as such, no such study exists.

193 A. E. O'Hare & O. B. Eden, Bleeding Disorders and Non-Accidental Injury, 59 Archives of Disease in Childhood 860, 863 (1984); P.G. Scimeca et al., Suspicion of Child Abuse Complicating the Diagnosis of Bleeding Disorders, 13 J. Pediatric Hematology/ Oncology 179 (1996).

194 Jami Jackson et al., Challenges in the Evaluation for Possible Abuse: Presentations of Congenital Bleeding Disorders in Childhood, 36 Child Abuse & Neglect 127, 131 (2012).

195 Id. at 130.

196 Id. at 131.

197 Id. at 133.

198 Id. at 131.

199 Id.

200 Id.

201 See Francesco Rodeghiero et al., The Discriminant Power of Bleeding History for the Diagnosis of Type 1 von Willebrand Disease: An International, Multicenter Study, 3 J. Thrombosis & Haemostasis 2619, 2619-20 (2005).

202 Id. at 2624.

203 See Jackson, supra note 193, at 127.

204 See Carpenter, supra note 163, at 1360-61; Jackson, supra note 167, at 128.

205 See Jackson, supra note 193, at 127-28.

206 Id.

207 Id. at 128.

208 See Carpenter, supra note 163, at 1368; Jackson, supra note 193, at 127-28.

209 See Oxford, supra note 18.

210 See Gardner, supra note 161, at 663.

211 Patrick D. Barnes et al., Infant Acute Life-Threatening Event-Dysphagic Choking Versus Nonaccidental Injury, 17 Seminars in Pediatric Neurology 7, 9-10 (2010).

212 See Clemetson, supra note 161, at 535.

213 Where A = a specific bleeding disorder; B = ICH due to bleeding disorder A; P = Probability/Prevalence; and PTP = Pre-test Probability.

214 See Metjain, supra note 190, at 918, 922; W.L. Nichols et al., Von Willebrand Disease (VWD): Evidence-based Diagnosis and Management Guidelines, the National Heart, Lung, and Blood Institute (NHLBI) Expert Panel Reprot (USA), 14 Haemophilia 171, 179 (2008).

215 Id. at 921.

216 "Parenchymal" bleeding refers to bleeding within the brain matter itself, not outside it like SDH, subarachnoid hemorrhage or epidural hemorrhage.

217 Carpenter et al., Technical Report: Evaluating for Suspected Child Abuse: Conditions that Predispose to Bleeding, Pediatrics (In Press). Used with permission.

218 See Carpenter, supra note 163, at 1360. Due to the method by which these forms of hemophilia are inherited, severe hemophilia is much more common in males than females.

219 See Table 1.

220 W.L. Nichols et al., Von Willebrand Disease (VWD): Evidence-Based Diagnosis and Management Guidelines, the National Heart, Lung, and Blood Institute (NHLBI) Expert Panel Report (USA), 14 Haemophilia 171, 171 (2008).

221 Id. at 197.

222 Id. at 172.

223 Id. at 179.

224 Id. at 181.

225 Id. at 179-80.

226 U.S. Dep't of Health and Human Serv., National Inst. of Health, National Heart Lung and Blood Inst., The Diagnosis, Evaluation, and Management of von Willebrand Disease 2, 32, 41, 49-50 (2007) (stating that repeat testing for VWD may be required because VWF levels may be artificially elevated in times of critical illness). Testing for VWD includes von Willebrand factor (VWF) levels, VWF activity, and Factor 8 levels.

227 Id. at 32. Bleeding symptoms of VWD are generally mild; therefore, it is reasonably assumed by clinicians that there are people with low VWF levels who have not come to medical attention.

228 Id. at 31.

229 Id. at 16. Individuals with bleeding symptoms and VWF levels between 30-50% currently pose a diagnostic dilemma for clinicians.

230 Id. at 1.

231 J. E. Sadler et al., Impact, Diagnosis and Treatment of von Willebrand Disease, 84 J. Thrombosis & Haemostasis 160, 164 (2000).

232 Walid S. Almaani & Abdulla S. Awidi, Spontaneous Intracranial Hemorrhage Secondary to von Willebrand Disease, 26 Surgical Neurology 457, 458 (1986); Kazuo Mizoi et al., Intracranial Hemorrhage Secondary to von Willebrand's Disease and Trauma, 22 Surgical Neurology 495, 498 (1984).

233 Id.

234 See Nichols, supra note 218, at 186-87.

235 See Almaani, supra 230, at 457.

236 Id. at 458.

237 See Mizoi, supra note 230, at 495-98; O. Ziv & M. V. Ragni, Bleeding Manifestations in Males with von Willebrand Disease, 10 Haemophilia 162, 162-68 (2004); T. Delangre et al., Hematome Intracerebral du a Une Forme Attenuee de Maladie de von Willebrand, 15 La Presse Medicale 1240, 1240-41 (1986); Robert Diecidue et al., Post-Traumatic Hemorrhage in a Patient with Previously Undiagnosed von Willebrand's Disease, 58 J. Oral Maxillofacial Surgery 332, 332-36 (2000); Kristine Slam et al., Common Bleeding Disorders: A Potential Catastrophe for the Trauma Victim. Therapeutic Recommendations for the Treatment of von Willebrand's Disease, 64 J. Trauma 1373, 1373-75 (2008).

238 See Mizoi, supra note 230, at 495.

239 See Martha E. Laposata & Michael Laposata, Children with Signs of Abuse. When is it Not Child Abuse?, 123 Am. J. Clinical Pathology (Supp. 1) 119, 121 (2005).

240 Arne Stray-Pedersen et al., An Infant with Subdural Hematoma and Retinal Hemorrhages: Does von Willebrand Disease Explain the Findings?, 7 Forensic Sci. Med. Pathology 37, 41 (2011).

241 See Mizoi et al., supra note 230; Slam et al., supra note 235. Additionally, the child with documented mild VWD and accidental head trauma was a four year old who suffered an accidental fall with a resulting large subdural hematoma that required surgical drainage. Mizoi et al., supra note 230, at 498. This clinical picture is significantly different from the vast majority of child victims of AHT where the children are less than one year old, immobile, often with no external signs of impact to the head, and with thin, bilateral convexity subdural hematomas.

242 Catherine P. M. Hayward et al., Congenital Platelet Disorders: Overview of Their Mechanisms, Diagnostic Evaluation and Treatment, 12 Haemophilia (Supp. 3) 128, 129 (2006).

243 Id. at 132; Otobia Dimson et al., Hermansky-Pudlak Syndrome, 16 Pediatric Dermatology 475, 475-77 (1999).

244 Emmanuel J. Favaloro, Clinical Utility of the PFA-100, 34 Seminars Thrombosis Hemostasis 709, 709-710 (2008); Hayward et al., supra note 240, at 133. The more specific platelet testing would assess platelet aggregation and secretion. Occasionally, electron microscopic examination or genetic testing is necessary to confirm the diagnosis.

245 See Hayward et al., supra note 240, at 132.

246 Id. at 130.

247 Id.

248 Laposata, supra note 237, at 120.

249 See Kzuo Mizoi et al., supra note 230; Slam et al., supra note 200; Joseph Broderick et al., Stroke, 38 Am. Heart Ass'n J. 2001-23 (2007), available at http://stroke.ahajournals.org/content/38/6/2001.full.

250 Giovanni Di Minno et al., Glanzmann's Thrombasthenia (Defective Platelet Integrin aIIb-#3): Proposals for Management Between Evidence and Open Issues, 102 Thrombosis Hematosis 1157, 1158 (2009); Jayanthi Alamelu & Ri Liesner, Modern Management of Severe Platelet Function Disorders, 149 Brit. J. Haematology 813, 813 (2010).

251 Di Minno et al., supra note 248, at 1157-1158; Amaelu & Liesner, supra note 248, at 813.

252 See Hayward et al., supra note 240, at 133; Favaloro, supra note 242, at 709.

253 See Oxford, supra note 18.

254 See Bob Phillips et al., Oxford Centre for Evidence-Based Medicine - Levels of Evidence (March 2009), CEBM, http://www.cebm.net/index.aspx?o=1025 (last edited Jan. 29, 2013).

255 See, e.g., Acharya et al., supra note 165.

256 Isaac Newton, Philosophiae Naturalis Principia Mathematica (London, S. Pepys, Reg. Soc. Praeses 1686), available at http://www.gutenberg.org/files/28233/28233-pdf.

257 See, e.g., Benjamin Crowell, Mechanics 14 (2012), available at http://www.lightandmatter.com/me.pdf.

258 The American Heritage Science Dictionary (Joseph P. Pickett et al. eds., 2005).

259 Susan Margulies & Brittany Coats, Biomechanics of Head Trauma in Infants and Young Children, in Child Abuse and Neglect: Diagnosis, Treatment and Evidence 359, 359 (Carole Jenny ed., 2011).

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262 Id.

263 Nicole G. Ibrahim et al., Physiological and Pathological Responses to Head Rotations in Toddler Piglets, 27 J. Neurotrauma 1021, 1021 (2010).

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265 See Carole Jenny, Child Abuse and Neglect: Diagnosis, Treatment and Evidence (Elsevier Saunders 2011).

266 Narang, supra note 10, at 542.

267 See Margulies & Coats, supra note 257, at 359, 362 (citing to works published earlier by Thomas A. Gennarelli and Lawrence E. Thibault).

268 Ann-Christine Duhaime et al., The Shaken Baby Syndrome: A Clinical, Pathological, and Biomechanical Study, 66 J. Neurosurgery 409, 409 (1987).

269 C.Z. Cory & M.D. Jones, Can Shaking Alone Cause Fatal Brain Injury? A Biomechanical Assessment of the Duhaime Shaken Baby Syndrome Model, 43 Med. Sci. & Law 317, 317 (2003). **161**

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271 Id. at 317.

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273 D.R. Wolfson et al., Rigid-Body Modeling of Shaken Baby Syndrome, 219 Proc. Inst. Mechanical Engineering (Part H: J. Engineering Med.) 63, 66 (2005).

274 Stephanie A. Eucker et al., Physiological and Histopathological Responses Following Closed Rotational Head Injury Depend on Direction of Head Motion, 227 Experimental Neurology 79, 85 (2011).

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280 Sébastien Roth et al., Finite Element Analysis of Impact and Shaking Inflicted to a Child, 121 Int'l J. Legal Med. 223, 223-28 (2007).

281 Id. at 224.

282 Id. at 227; see also H. Maxeiner, Detection of Ruptured Cerebral Bridging Veins at Autopsy, 89 Forensic Sci. Int'l 103, 103-10 (1997) (Injury to the bridging veins is believed to be the cause of subdural hematomas in AHT).

283 Limitations of the data in this study include the fact that the authors of the studies did not provide properties for vessels, and modeled the CSF as a solid material. Additionally, they modeled the vein as an elastic spring, which is not rate dependent.

284 Christopher Neil Morison, The Dynamics of Shaken Baby Syndrome (June 2002) (unpublished Ph.D. dissertation, University of Birmingham) (on file with the University of Birmingham Research Archive).

285 Id. at 61.

286 Id. at 82.

287 John W. Finnie et al., Diffuse Neuronal Perikaryal Amyloid Precursor Protein Immunoreactivity in an Ovine Model of Non-Accidental Head Injury (The Shaken Baby Syndrome), 17 J. Clinical Neuroscience 237 (2010).

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289 Id.

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292 Catherine Adamsbaum et al., Abusive Head Trauma: Judicial Admissions Highlight Violent and Repetitive Shaking, 126 Pediatrics 546, 546-55 (2010).

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294 Id.

295 Id.

296 Prange, supra note 275, at 149 (emphasis added).

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298 Faris A. Bandak, Shaken Baby Syndrome: A Biomechanical Analysis of Injury Mechanisms, 151 Forensic Sci. Int'l 71, 71-79 (2005).

299 Id. at 78.

300 Id. at 71.

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303 Bandak, supra note 296, at 76.

304 Id. at 76-78.

305 Id. at 77. See also J. Matthews Duncan, Laboratory Note: On the Tensile Strength of the Fresh Adult Fetus, 2 Br. Med. J. 763, 763-64 (1874); Randal P. Ching et al., Tensile Mechanics of the Developing Cervical Spine, 45 Stapp Car Crash 329, 329-36 (2001); R. Mayer et al., Pediatric Tensile Neck Strength Characteristics Using a Caprine Model, Injury Biomechanics Research, Proceedings of the 27th International Workshop on Human Subjects Biomechanics 87-92 (1999).

306 Bandak, supra note 296, at 79.

307 See Randal P. Ching, supra note 303; R. Mayer et al., supra note 303.

308 N. Rangarajan & T. Shams, Letter to the Editor, 164 Forensic Sci. Int'l 280, 281 (2006).

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311 Laura K. Brennan et al., Neck Injuries in Young Pediatric Homicide Victims, 3 J. Neurosurgery Pediatrics 232, 235 (2009) (finding that 71% of children dying of AHT had injuries to the cervical spine or cord).

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313 Susan Margulies et al., Letter to the Editor: Shaken Baby Syndrome: A Flawed Biomechanical Analysis, 164 Forensic Sci. Int'l 278, 278 (2006).

314 Id. at 278.

315 Id. at 279.

316 Rangarajan & Shams, supra note 306, at 280.

317 Faris A. Bandak, Response to the Letter to the Editor, 164 Forensic Sci. Int'l 282, 282 (2006).

318 Id. at 282.

319 This author does not understand what this statement means in a rigorous, mathematical sense. However, because Bandak does not specify even the length or frequency of shaking used in his computations, no scientist will be able to replicate his result.

320 See Cavazos v. Smith, 565 U.S. 1, 5 (2011) (per curiam) (Ginsburg, J., dissenting).

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322 Timothy J. Kriewall, Structural, Mechanical, and Material Properties of Fetal Cranial Bone, 142 Am. J. Obstetrics Gynecology 707, 707 (1982); Gregg K. McPherson & Timothy J. Kriewall, The Elastic Modulus of Fetal Cranial Bone: a First Step Towards Understanding of the Biomechanics of Fetal Head Molding, 13 J. Biomechanics 9, 14 (1980); Brittany Coats & Susan S. Margulies, Material Properties of Human Infant Skull and Suture at High Rates, 23 J. Neurotrauma 1222, 1222-32 (2006).

323 See Coats & Margulies, supra note 320.

324 Id.

325 See Kriewall, supra note 320. The "elastic modulus" is a ratio of stress to strain, and is constant for any uniform material. The smaller the elastic modulus, the more readily deformable it is.

326 See Coats & Margulies, supra note 320.

327 Id.

328 The "suture" is the area of incomplete fusion of the cranial bone. It permits growth of the cranial bone and the intracranial structures. Neil K. Kaneshiro, Cranial Sutures, University of Maryland Medical Center Medical Encyclopedia (Feb. 21, 2013), http:// umm.edu/ health/medical/ency/articles/cranial-sutures.

329 See Coats & Margulies, supra note 320.

330 See W. Weber, Experimentelle Untersuchungen zu Schädelbruchverletzungen des Säuglings [Experimental Study of Skull Fractures in Infants], 92 Zeitschrift für Rechtsmedizin 87 (1984); W. Weber, ZurBiomechanischem Fragilität des Säuglingsschädels [Biomechanical Fragility of Skull Fractures in Infants], 94 Zeitschrift für Rechtsmedizin 93 (1985).

331 W. Weber, Experimental Study of Skull Fractures in Infants, supra note 328 at 90. Five cadavers were dropped on each type of flooring surface. Id.

332 Id. at 89-91.

333 Id. at 91.

334 Id. at 90-91.

335 W. Weber, Biomechanical Fragility of Skull Fractures in Infants, supra note 328, at 100.

336 Id. at 94.

337 Id. at 94-95.

338 Id. at 95.

339 See Richard G. Snyder et al., Highway Safety Research Inst., Univ. of Mich., Study of Impact Tolerance Through Free-Fall Investigations: Final Report (1977).

340 Id. at 11-14, 38.

341 Id. at 38, App'x A.

342 Id. at 20, 23-32, 76, 126.

343 Id. at 78.

344 Id.

345 Id. at 81.

346 Id. at 120-21. In another study, Snyder et al. abstracted 954 cases of children aged 1-12 years in which fall height, severity of injury, and fall surface (steel or concrete) were known from a database maintained by the FAA of 31,530 adult and child free-falls (fatal and non-fatal). See Richard G. Snyder, Highway Safety Research Inst., Univ. of Mich., Impact Tolerances of Infants and Children in Free-Falls (1970). Of those 954 cases, the authors took 34 cases in which biometric testing of the patient and full characterization of the fall and landing zone could be performed. Id. The LD50 (the height at which roughly 50% die) occurred between 41-50 feet. Id. According to Snyder's data, with lower free falls came lower fatality rates and lower rates of serious or critical injuries. Id. There were no deaths in falls of 0-5 foot free falls onto steel or concrete in this series. Id.

347 Gina E. Bertocci et al., Using Test Dummy Experiments to Investigate Pediatric Injury Risk in Simulated Short-Distance Falls, 157 Archives Pediatrics Adolescent Med. 480, 481-82 (2003).

348 Id. at 482-83.

349 Brittany Coats et al., Parametric Study of Head Impact in the Infant, 51 Stapp Car Crash J. 1, 1-2 (2007).

350 Id. at 5.

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352 Id. at 2, 8, 11.

353 See Lyons & Oates, supra note 64, at 126-27; Helfer, supra note 61, at 534-35; Nimityongskul, supra note 68, at 185-86.

354 Prange, supra note 275, at 144-45.

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358 Angela K. Thompson et al., Assessment of Head Injury Risk Associated with Feet-First Free Falls in 12 Month-Old Children Using an Anthropomorphic Test Device, 66 J. Trauma Infection & Critical Care 1019, 1020 (2009).

359 Id.

360 Id. at 1028.

361 Ernest Deemer et al., Influence of Wet Surfaces and Fall Height on Pediatric Injury Risk in Feet-First Freefalls as Predicted Using a Test Dummy, 27 Med. Engineering & Physics 31, 38 (2005).

362 See Thompson, supra note 98, at 144. Parents or guardians of children selected for case study had the option to allow the investigators to: 1) review the child's medical records; 2) interview the caregiver and review the child's medical records; or 3) investigate the fall scene at the home, interview the caregiver, and review the child's medical records. Id.

363 Id.

364 Id. at 148.

365 Id.

366 Nagarajan Rangarajan et al., Finite Element Model of Ocular Injury in Abusive Head Trauma, 13 J. Am. Ass'n for Pediatric Ophthalmology & Strabismus 364, 365 (2009).

367 Id. at 368.

368 Id.

369 See Sandeep Narang, A Daubert Analysis of Abusive Head Trauma/Shaken Baby Syndrome, 11 Hous. J. Health L. & Pol'y 505, 548-58 (2011).

370 Steven Alex Hans et al., A Finite Element Infant Eye Model to Investigate Retinal Forces in Shaken Baby Syndrome, 247 Graefe's Archive for Clinical & Experimental Ophthalmology 561, 567-68 (2009).

371 Id. at 568.

372 Id. at 568-70.

373 See Susan Margulies et al., What Can We Learn from Computational Model Studies of the Eye?, 13 J. Am. Ass'n for Pediatric Ophthalmology & Strabismus 332, 332 (2009).

374 Id.

375 Id.

376 Brittany Coats et al., Ocular Hemorrhages in Neonatal Porcine Eyes from Single, Rapid Rotational Events, 51 Investigative Opthalmology & Visual Sci. 4792, 4792 (2010).

377 Id. at 4793.

378 Id. at 4794.

379 Finnie, supra note 285, at 237, 239.

380 Oxygen is carried in the blood from the lungs to the body and organs by two main mechanisms. The vast majority of oxygen in the blood is bound to hemoglobin (a protein in red blood cells), with a smaller percentage of oxygen actually dissolved in the blood.

381 Terry R. Des Jardins, Cardiopulmonary Anatomy & Physiology: Essentials for Respiratory Care 211-244 (2002).

382 Id.

383 J.F. Geddes et al., Dural Haemorrhage in Non-Traumatic Infant Deaths: Does it Explain the Bleeding in 'Shaken Baby Syndrome'?, 29 Neuropathology & Applied Neurobiology 14, 18-19 (2003).

384 See D. G. Talbert, Paroxysmal Cough Injury, Vascular Rupture and 'Shaken Baby Syndrome', 64 Med. Hypotheses 8, 8-13 (2005); D. G. Talbert, Dysphagia as a Risk Factor for Sudden Unexplained Death in Infancy, 67 Med. Hypotheses 786 (2006); J.F. Geddes & D.G. Talbert, Paroxysmal Coughing, Subdural and Retinal Bleeding: A Computer Modelling Approach, 32 Neuropathology & Applied Neurobiology 625 (2006); Patrick D. Barnes et al., Infant Acute Life-Threatening Event--Dysphagic Choking Versus Nonaccidental Injury, 17 Seminars in Pediatric Nurology 7 (2010).

385 Evan W. Matshes et al., Shaken Infants Die of Neck Trauma, Not of Brain Trauma, 1 Acad. Forensic Pathology 82, 83-86 (2011).

386 See J.F. Geddes et al., Neuropathology of Inflicted Head Injury in Children I. Patterns of Brain Damage, 124 Brain 1290 (2001); J.F. Geddes et al., Neuropathology of Inflicted Head Injury in Children II. Microscopic Brain Injury in Infants, 124 Brain 1299 (2001); J.F. Geddes et al., supra note 381, at 14.

387 Geddes et al., supra note 384, at 1290.

388 Geddes et al., supra note 384, at 1299.

389 See Geddes et al., supra note 381.

390 See Geddes et al., supra note 384.

391 Id. at 1290.

392 The authors identified AHT cases either: 1) by confession; 2) by criminal conviction, with or without the presence of extracranial injuries on the child; 3) by cases without conviction in which unexplained injuries to the rest of the child's body, in addition to head injury, were present; or 4) by cases in which there was a "major discrepancy" between the injury explanation given by the caregiver and "significant injuries" or "the history was developmentally incompatible" with the injury. Id. at 1290-91.

393 See, e.g., Linda Ewing-Cobbs et al., Neuroimaging, Physical, and Developmental Findings After Inflicted and Noninflicted Traumatic Brain Injury in Young Children, 102 Pediatrics 300 (1998); K.W. Feldman et al., The Cause of Infant and Toddler Subdural Hemorrhage: A Prospective Study, 108 Pediatrics 636 (2001); K.P. Hymel et al., Mechanisms, Clinical Presentations, Injuries, and Outcomes from Inflicted Versus Noninflicted Head Trauma During Infancy: Results of a Prospective, Multicentered, Comparative Study, 119 Pediatrics 922 (2007).

394 Geddes et al., supra note 384, at 1291-94.

395 Id. at 1291. Although one case had only a clinical history available for review, the investigators found "sufficient detail to merit [the case's] inclusion in the study." Id.

396 Id.

397 Id. at 1291-92.

398 Id.

399 Id. at 1292. The authors note that the "thin film" designation was used in post-mortem reports in which the SDHs found were "trivial in terms of quantity of blood." Id.

400 Id. at 1294.

401 Id.

402 Id.

403 Id. It is important to note that if the author examined the other five subjects without SDH and either four or five of them had RH, then the relationship would no longer be significant by chi square testing (p=0.068 or p=0.209). By not examining all of the eyes of their "control" group, they falsely report significance when the relationship may not be true.

404 Id. at 1295-96.

405 Id. at 1294.

406 Id. at 1292.

407 Id. at 1295-96.

408 Id. at 1294.

409 Id.

410 Id. at 1291-92.

411 Geddes et al., supra note 384, at 1299-1300.

412 Id.

413 "Histology" means the microscopic evaluation of tissue at the cellular level. Histology, Merriam-Webster, http://www.merriam-webster.com/dictionary/histology (last visited Aug. 4, 2013).

414 Geddes et al., supra note 384, at 1300-01.

415 Id. at 1299-1302.

416 See Geddes et al., supra note 384, at 1294; Michael W. Johnson et al., Axonal Injury in Young Pediatric Head Trauma: A Comparison Study of ß-Amyloid Precursor Protein (ßAPP) Immunohistochemical Staining in Traumatic and Nontraumatic Deaths, 56 J. Forensic Scis. 1198, 1198 (2011).

417 Geddes et al., supra note 384, at 1300.

418 Id.

419 Id. at 1300-01.

420 Id. at 1302.

421 Id.

422 See id.

423 Id. at 1304.

424 See Geddes et al., supra note 381, at 15.

425 Id.

426 See id.

427 Id. The authors labeled the comparison cases as "cases of classical 'shaken baby syndrome.'" Id.

428 See id.

429 Id. at 15, 17-18.

430 Id. at 15.

431 Id.

432 Id.

433 Id.

434 Id.

435 Id.

436 See id. at 15, 17-18.

437 Id. at 19.

438 Id. at 14.

439 See id. at 17.

440 Id. at 19-20.

441 Id. at 19.

442 Id. at 19-20.

443 Johnson et al., supra note 90, at 1199-1200.

444 Id. at 1199.

445 Id.

446 Id. at 1201.

447 Id. at 1198.

448 Geddes et al., supra note 384, at 1290-91; Geddes et al., supra note 384, at 1299.

449 See A.C. Duhaime et al., Head Injury in Very Young Children: Mechanisms, Injury Types, and Opthalmologic Findings in 100 Hospitalized Patients Younger than 2 Years of Age, 2 Pediatrics 179 (1992); Linda Ewing-Cobbs et al., Neuroimaging, Physical, and Developmental Findings After Inflicted and Noninflicted Traumatic Brain Injury in Young Children, 102 Pediatrics 300 (1998); K.W. Feldman et al., The Cause of Infant and Toddler Subdural Hemorrhage: A Prospective Study, 108 Pediatrics 636 (2001); K.P. Hymel et al., Mechanisms, Clinical Presentations, Injuries, and Outcomes from Inflicted Versus Noninflicted Head Trauma During Infancy: Results of a Prospective, Multicentered, Comparative Study, 119 Pediatrics 922 (2007); S. Maguire et al., Which Clinical Features Distinguish Inflicted from Non-Inflicted Brain Injury? A Systematic Review, 94 Archives of Disease in Childhood 860, 860-67 (2009).

450 W. Squier, Shaken Baby Syndrome: The Quest for Evidence, 50 Dev. Med. Child 10, 10-14 (2008).

451 Patrick D. Barnes, Imaging of Nonaccidental Injury and the Mimics: Issues and Controversies in the Era of Evidence-Based Medicine, 49 Radiologic Clinics N. Am., 205, 205-29.

452 "Meningitis" indicates an infection or inflammation of the meninges (the outer covering of the brain). Rodrigo Hasbun, Meningitis, Medscape, http://www.emedicine.medscape.com/article/232915-overview (last visited Aug. 4, 2013).

453 "Sepsis" is a potentially deadly condition characterized by whole body inflammation and organ dysfunction, usually caused by an infection. Shankar Santhanam & Russell W. Steele, Pedriatric Sepsis, Medscape, http:// www.emedicine.medscape.com/ article/972559-overview#a0104 (last visisted Aug. 4, 2013).

454 "Pyelonephritis" means infection or inflammation of the kidney. Robert W. Tolan et al., Pediatric Pyelonephritis, Medscape, http:// www.emedicine.medscape.com/article/968028-overview (last visited Aug. 4, 2013).

455 "Intradural" hemorrhage means inside the dura, or not macroscopically apparent on the surface of the brain. See Geddes et al., supra note 433, at 15.

456 See William H. Chase, An Anatomical Study of Subdural Haemorrhage Associated with Tentorial Splitting in the Newborn, 51 Surgery Gynecology Obstetrics 31 (1930). Philip Schwartz & Eardely L. Holland, Birth Injuries of the Newborn: Morphology, Pathogenesis, Clinical Pathology and Prevention (S. Karger ed., 1961). Schwartz reports that intracranial hemorrhage was first described in 1804. Id. Schwartz outlines the continued study of falcine and tentoral hemorrhage (different sections of the dura) in newborns for over 100 years. Id.

457 Id.

458 See Geddes et al., supra note 381.

459 See Schwartz, supra note 454.

460 Id. at 38.

461 Jean Cruveilhier, Paper presented at Confer. a l'occasion de la disrtibution des prix aux eleves sages-femmes de las Maison d'Accouchement del Paris, (June 23, 1831) (Fr.).

462 See Schwartz, supra note 454.

463 See V. J. Rooks et al., Prevalence and Evolution of Intracranial Hemorrhage in Asymptomatic Term Infants, 29 Am. J. Neuroradiology 1082 (2008); E. H. Whitby et al., Frequency and Natural History of Subdural Haemorrhages in Babies and Relation to Obstetric Factors, 362 Lancet 846 (2003).

464 See Cruveilhier, supra note 459; Schwartz, supra, note 454. Of note, when reporting a P-value to three decimal points, any value below 0.0005 is simply reported as 0.000. Thus, a 0.0006 would be reported as 0.001, but 0.0003 would be 0.000.

465 In a subsequent study, Dr. Geddes' colleagues attempted to demonstrate hypoxia-associated SDHs, but utilized a cohort of patients that were fetuses (as premature as twenty-six weeks) and young neonates (the oldest being only nineteen days). See Marta C. Cohen & Irene Scheimberg, Evidence of Occurrence of Intradural and Subdural Hemorrhage in the Perinatal and Neonatal Period in the Context of Hypoxic Ischemic Encephalopathy: an Observational Study from Two Referral Institutions in the United Kingdom, 12 Pediatric Dev. Pathology 169 (2009).

466 Karim T. Rafaat et al., Cranial Computed Tomographic Findings in a Large Group of Children with Drowning: Diagnostic, Prognostic, and Forensic Implications, 9 Pediatric Critical Care Med. 567 (2008).

467 Id.

468 The arrival at the upper limit of 2% is analogous to a "margin of error" in statistics. In statistical analysis, the 2% is the largest outside chance that these findings would be present if the study were large enough to include all possible cases of drowning.

469 Steven B. Taylor et al., Central Nervous System Anoxic-Ischemic Insult in Children Due to Near-Drowning, 156 Pediatric Radiology 641 (1985).

470 Roger W. Byard et al., Lack of Evidence for a Causal Relationship Between Hypoxic-Ischemic Encephalopathy and Subdural Hemorrhage in Fetal Life, Infancy, and Early Childhood, 10 Pediatric Dev. Pathology 348, 348 (2007).

471 M. Hurley et al., Is There a Causal Relationship Between the Hypoxia-Ischaemia Associated with Cardiorespiratory Arrest and Subdural Haematomas? An Observational Study, 83 Brit. J. Radiology 736, 736-37 (2010).

472 Raymond D. Pitetti et al., Prevalence of Retinal Hemorrhages and Child Abuse in Children Who Present with an Apparent Life-Threatening Event, 110 Pediatrics 557 (2002).

473 Id.

474 Amy Odom et al., Prevalence of Retinal Hemorrhages in Pediatric Patients After In-Hospital Cardiopulmonary Resuscitation: A Prospective Study, 99 Pediatrics 861 (1997).

475 Id.

476 Charanjit Kaur et al., Early Response of Neurons and Glial Cells to Hypoxia in the Retina, 47 Investigative Ophthalmology Visual Sci. 1126 (2006) (no RHs in rats with hypoxia of the retina); Taiji Nagaoka et al., The Effect of Nitric Oxide on Retinal Blood Flow During Hypoxia in Cats, 43 Investigative Ophthalmology Visual Sci. 3037 (2002) (decreased retinal blood flow leads to vessel dilation but no RHs in cats).

477 See Talbert, supra note 382. Talbert initially proposed that coordinated coughs could cause a cascade of increased systemic arterial pressure that was beyond the threshold of cerebral blood vessels. Id. This increased blood pressure would then lead to SDH and RH. Id. Talbert identified pertussis (whooping cough) as a "natural experiment" of this phenomenon. Id.

478 See Geddes & Talbert, supra note 382; Findley et al., supra note 3; see also Mudher Al-Adnani et al., Gastroesophageal Reflux Disease and Sudden Infant Death: Mechanisms Behind an Under-Recognized Association, 14 Pediatric Dev. Pathology 53 (2010).

479 See Talbert, supra note 382.

480 D.G. Talbert, Pyloric Stenosis as Cause of a Venous Hypertensive Syndrome Mimicking True Shaken Baby Syndrome, 1 J. Trauma Treatment 1, 5, 8 (2012).

481 D.G. Talbert, Cyclic Vomiting Syndrome: Contribution to Dysphagic Infant Death, 73 Med. 473, 474-77 (2009).

482 See Geddes & Talbert, supra note 382, at 627-29.

483 Id. at 629.

484 See Barnes et al., supra note 382, at 7, 9-10.

485 Id. at 7.

486 Id. at 7-8.

487 Id. at 8.

488 Id.

489 Id. at 10.

490 Id.

491 See Matshes, supra note 383.

492 Id.

493 Id.

494 Id.

495 Id. at 84.

496 Id.

497 Id. at 88.

498 See Geddes & Talbert, supra note 382, at 629.

499 Id. at 630.

500 Thomas v. State, No. 03-07-00646-CR, 2009 WL 1364348, at *1 (Tex. App.--Austin May 14, 2009).

501 Christopher S. Greeley, Letter to the Editor, 17 Seminars Pediatric Neurology 275, 277 (2010).

502 John Galaznik et al., Reply to Greeley et al, 17 Seminars Pediatric Neurology 279 (2010).

503 While the authors reported an acute rib fracture was from CPR, the vignette, as presented in the medical literature, did not mention the presence of a healing rib fracture. See Barnes, supra note 382, at 10. A review of the court transcripts indicates that at least one of the co-authors was clearly aware of the rib fracture. Thomas, 2009 WL 1364348 at *4-5.

504 See Talbert, supra note 382.

505 Div. of Bacterial Diseases, Nat'l Ctr. for Immunization & Respiratory Diseases, Pertussis: Disease Specifics, CDC (May 7, 2012), http:// www.cdc.gov/pertussis/clinical/disease-specifics.html.

506 James D. Cherry, Pertussis in the Preantibiotic and Prevaccine Era, with Emphasis on Adult Pertussis, Clinical Infectious Diseases (Supplement), 107, 107 (1999).

507 Id. at 109.

508 J.N. Marshall, Aphasia and Cerebral Haemorrhage Complicating Whooping-Cough, 23 Glasgow Med. J. 24 (1885). While this is often cited as a case with SDH, the manuscript actually indicates that there was not an SDH, as is often contended there was.

509 Am. Acad. of Pediatrics: Pertussis, in Red Book: 2003 Rep. of the Committee of Infectious Diseases 472 (Pickering ed., 2003), available at http://www.aapredbook.aappublications.org/cgi/content/full/2003/1/3.9.

510 Id.

511 Id.

512 Ana I. Curcoy et al., Is Pertussis in Infants a Potential Cause of Retinal Haemorrhages?, 97 Arch. Dis. Child. 239 (2012).

513 Michael Goldman et al., Severe Cough and Retinal Hemorrhage in Infants and Young Children, 148 J. Pediatrics 835. 836 (2006).

514 Sandra Herr et al., Does Valsalva Retinopathy Occur in Infants? An Initial Investigation in Infants with Vomiting Caused by Pyloric Stenosis, 113 Pediatrics 1658 (2004).

515 See generally Matshes, supra note 383. In fact, some cases were included if neck hyperflexion/hyperextension was merely suspected. Id. at 83.

516 Id. at 87.

517 See Bob Phillips et al., Oxford Centre for Evidence-based Medicine Levels of Evidence (2009), available at http://www.cebm.net/index.aspx?o=4590.

518 Learned Hand, Historical and Practical Considerations Regarding Expert Testimony, 15 Harv. L. Rev., 40, 50 (1901) (emphasis added).

519 In his writings, Judge Hand argued for the existence of "competent tribunal" or "a single expert," not called by either side, "who have possessed themselves the specialized experience" and "trained powers of observation," to "advise the jury of the general propositions applicable to the case." See id. at 55-56.

520 Frye v. United States, 293 F. 1013 (D.C. Cir. 1923).

521 The Frye "general acceptance" test remains the rule governing admissibility of scientific expert testimony in several states, including California, Florida, Illinois, Kansas, Maryland, Minnesota, New Jersey, New York, Pennsylvania, and Washington. Alice Lustre, Post-Daubert Standards of Admissibility of Scientific and Other Expert Evidence in State Courts, 90 A.L.R. 5th 453, §§ 28-43 (2001).

522 Fed. R. Evid. 702.

523 Daubert v. Merrell Dow Pharmaceuticals, Inc., 509 U.S. 579, 590 (1993).

524 Id. at 590-91, n.9.

525 1) Falsifiability; 2) Peer review and publication; 3) Known or potential rate of error; and, 4) General acceptance. Id. at 593-94.

526 General Electric Co. v. Joiner, 522 U.S. 136 (1997).

527 Kumho Tire Co. v. Carmichael, 526 U.S. 137 (1999).

528 See Joiner, supra note 524, at 146.

529 See Kumho, supra note 525, at 157.

530 While the gatekeeper must also assess relevance and potential 403(b) objections, our analysis will focus on these three legal questions.

531 See United States v. Addison, 498 F.2d 741, 744 (1974) (The Court stated, "scientific proof may in some instances assume a posture of mystic infallibility in the eyes of a jury of laymen."). See also J.W. Strong, Language and Logic in Expert Testimony: Limiting Expert Testimony by Restrictions of Function, Liability, and Form, 71 Or. L. Rev. 349, 367, n.81 (1992) ("There is virtual unanimity among courts and commentators that evidence perceived by jurors to be 'scientific' in nature will have particularly persuasive effect"); Neil Vidmar & Shari S. Diamond, Juries and Expert Evidence, 66 Brook. L. Rev. 1121, 1125 (2000) (quoting an amicus brief filed on behalf of the defendant in Kumho Tire Co. Ltd. v. Carmichael, 526 U.S. 137 (1999), "[Because of the 'aura of infallibility'], even when jurors have a 'basis for questioning the expert's reliability [they] may be disinclined to do so.'").

532 See generally, Vidmar & Diamond, supra note 529, at 1140-49.

533 Edward Imwinkelried, Shaken Baby Syndrome: A Genuine Battle of the Scientific (and Non-Scientific) Experts, 46 Crim. L. Bull. 156, 186-87 (2010) (The author asserts that proponents of the syndrome primarily rest their support upon the "admissions by caretakers" in Caffey's 1974 article and on the "experience" of "forensic pathologists.").

534 Id. at 184.

535 Id. at 188.

536 Daubert, 509 U.S. at 590.

537 William Stead & John Starmer, Beyond Expert-Based Practice, Evidence-Based Medicine and the Changing Nature of Health Care: 2007 IOM Annual Meeting Summary 94 (Inst. of Med., Mark McClellan et al. eds., 2008).

538 Id.

539 See State v. Sanchez-Cruz, 33 P.3d 1037, 1042 (Or. Ct. App. 2001) (emphasis in original) (citing Jennings v. Baxter Healthcare Corp., 14 P. 3d 596, 606 (Or. 2000).

540 Brief of the Am. Med. Assoc. et al., as Amici Curiae Supporting Respondents at 4, Daubert v. Merrel Dow Pharmaceuticals, Inc., 951 F.2d 1128 (9th Cir. 1991) (No. 90-55397), 1993 WL 13006285 (emphasis added).

541 See Daubert, 509 U.S. at 590 (emphasis added).

542 See State v. McMullen, 900 A.2d 103, 114-15 (Del. Super. Ct. 2006).

543 See id. at 116-19; see generally McClellan et al., supra note 36, at 94. This is to be distinguished from "differential etiology," which is a "legal invention not used by physicians." John B. Wong et al., Fed Judicial Ctr., Reference Guide on Medical Testimony, in Reference Manual on Scientific Evidence 691 (3rd ed. 2011) available at http://books.nap.edu/openbook.php? record_id=13163&page=R1.

544 See Daubert, 509 U.S. at 590, 593.

545 Narang, supra note 10, at 539-40, 583.

546 See Committee on Identifying the Needs of the Forensic Sciences Community, Nat'l Research Council, Strengthening Forensic Science in the United States: A Path Forward 112 (2009), available at http:// www.nap.edu/catalog/12589.html.

547 See Findley et al., supra note 3, at 218-20.

548 Anectodal Definition, Merriam-Webster.com, http://www.merriam-webster.com/dictionary/anecdotal (defining "anecdotal evidence" as "based on or consisting of reports or observations of usually unscientific observers"). See David H. Kaye & David A. Freedman, Fed. Judicial Ctr., Reference Guide on Statistics, Reference Manual on Scientific Evidence 90-92 (2nd ed. 2000).

549 See Finnie et al., supra note 285.

550 See supra Part V, Biomechanics.

551 Catherine Adamsbaum et al., Abusive Head Trauma: Judicial Admissions Highlight Violent and Repetitive Shaking, 126 Pediatrics 553-54. (2010).

552 Suzanne P. Starling et al., Analysis of Perpetrator Admissions to Inflicted Traumatic Brain Injury in Children, 158 Archives Pediatric Adolescence Med. 457 (2004); Erica Bell et al., Abusive Head Trauma: A Perpetrator Confesses, 35 Child Abuse & Neglect 74-77 (2011).

553 Anchoring Bias in Decision-Making, ScienceDaily.com, http:// www.sciencedaily.com/articles/a/anchoring.htm ("During normal decision making, individuals anchor, or overly rely, on specific information or a specific value and then adjust to that value to account for other elements of the circumstance.").

554 See McMullen, 900 A.2d at 114 (stating, "Because the objectives, functions, subject matter and methodology, of hard science vary significantly from those of the discipline of clinical medicine, as distinguished from research or laboratory medicine, the hard science

techniques or methods that became the 'Daubert factors' generally are not appropriate for assessing the evidentiary reliability of a proffer of expert clinical medical testimony.").

555 See Daubert, 509 U.S. at 594-95 (emphasis added).

556 See Narang, supra note 10, at 583-84.

557 Edward Imwinkelried, The "Bases" of Expert Testimony: The Syllogistic Structure of Scientific Testimony, 67 N.C. L. Rev. 1, 2-3 (1988) (emphasis added); see also Hand, supra note 516, at 51-52 (describing expert testimony as the application of the "major premise" to the "minor premise").

558 See Breyer, supra note 8, at 4.

559 See Hamilton, supra note 4; Imwinkelried, supra note 555 at 14 n.105 (quoting State v. Hyatt, No. O6MJ-CR00016-02 (Mo. Cir. Ct. Nov. 6, 2007) ("[I]n an unpublished order, the trial judge found that the prosecution had not met its burden of proving that shaken baby syndrome is generally accepted in the scientific and medical circles.").

560 Sophia Gatowski et al., Asking the Gatekeepers: A National Survey of Judges on Judging Expert Evidence in a Post-Daubert World, 25 Law & Human Behavior 433, 444-47 (2001).

561 See Findley et al., supra note 3, at 286-87 (in discussing the scientific literature, the authors state, "[e]ven if the causes were accurately classified, however, this measure [the P-value] provides no indication of the strength of the correlation for it does not distinguish between weak correlations ... and strong ones.... Yet the strength of the correlation is precisely what is needed to satisfy fact finding requirements in criminal cases, which requires proof beyond a reasonable doubt. Statistical significance is necessary but not sufficient to support this evidentiary standard.") (emphasis added).

562 See Tuerkheimer, supra note 3, at 11.

563 Id. at 12.

564 Id. at 14.

565 See Molly Gena, Shaken Baby Syndrome: Medical Uncertainty Casts Doubt on Convictions, 3 Wisc. L. Rev. 701, 701-727 (2007); Matthew D. Ramsey, A Nuts and Bolts Approach to Litigating the Shaken Baby or Shaken Impact Syndrome, 188 Military L. Rev. 1, 1 (2006).

566 See Findley et al., supra note 3; Imwinkelried, supra note 555.

567 See Cavazos, 565 U.S. 132 (2011).

568 See Narang, supra note 10, at 596-627.

569 See generally id. at 541-60.

570 Id. at 576-88.

571 See Tuerkheimer, supra note 3, at 12-13 n.79 (quoting Patrick D. Barnes, Imaging of the Central Nervous System in Suspected or Alleged Nonaccidental Injury, Including the Mimics, 18 Topics Magnetic Resonance Imaging 53, 55 (2007).

572 See Findley et al., supra note 3, at 296.

573 See Tuerkheimer, supra note 3, at 12-13.

574 See Findley et al., supra note 3, at 286-88.

575 See Tuerkheimer, supra note 3, at 14.

576 See Findley et al., supra note 3, at 242.

577 Id. at 289.

578 Rebecca Crosier, Shaken Baby Syndrome, Shaken Baby Syndrome, http://neurowiki2012.wikispaces.com/Shaken+Baby+Syndrome. (discussing in Part 2.2 and 2.3 alternative causes of SDH and RH and the need for medical professionals to be careful in a diagnosis of SBS).

579 See Narang, supra note 10, at 548, 559, 571, 579, 595.

580 See Findley et al., supra note 3, at 303 ("In arguing admissibility under Daubert, moreover, it is unclear what Dr. Narang believes should be admitted. Evidence that some brain injuries in children are of traumatic origin, sometimes even intentionally inflicted? Evidence that subdural hematomas and retinal hemorrhages are seen in cases of inflicted abuse? Evidence that shaking can cause the triad and can lead to injury or death? Evidence that subdural hematomas and retinal hemorrhages are diagnostic of shaking or abuse in the absence of a major motor vehicle accident, fall from a multistory building or other proven alternative? Some of these questions are not controversial ....").

581 C. Hobbs et al., Subdural Haematoma and Effusion in Infancy: An Epidemiological Study, 90 Archives Disease Childhood 952, 954 (2005); Victoria Trenchs et al., Subdural Haematomas and Physical Abuse in the First Two Years of Life, 43 Pediatric Neurosurgery 352, 352-53, 356 (2007); Dimitra Tzioumi & R. Kim Oates, Subdural Hematomas in Children Under 2 Years. Accidental or Inflicted? A 10-Year Experience, 22 Child Abuse & Neglect 1105, 1106-07 (1998).

582 Kenneth W. Feldman et al., The Cause of Infant and Toddler Subdural Hemorrhage: A Prospective Study, 108 Pediatrics 636, 638 (2001) (finding that 59% of trauma SDHs were "intentional," but only 23% were "accidental'); Jakob Matschke et al., Nonaccidental Head Injury is the Most Common Cause of Subdural Bleeding in Infants < 1 Year of Age, 124 Pediatrics 1587, 1594 (2009) (finding that 93% of trauma SDHs were "non-accidental" and only 7% were "accidental"); Hobbs et al., supra note 579 at 953 (finding that 94% of trauma SDHs were "non-accidental" and only 6% were "accidental").

583 Ann-Christine Duhaime et al., Head Injury in Very Young Children: Mechanisms, Injury Types, and Ophthalmologic Findings in 100 Hospitalized Patients Younger than 2 Years of Age, 90 Pediatrics 179, 183 (1992); Kirsten Bechtel, et al., Characteristics that Distinguish Accidental from Abusive Injury in Hospitalized Young Children with Head Trauma, 114 Pediatrics 165, 165, 168 (2004); Matthieu Vinchon et al., Confessed Abuse Versus Witnessed Accidents in Infants: Comparison of Clinical, Radiological, and Ophthalmological Data in Corroborated Cases, 26 Child's Nervous Sys. 637, 638-39 (2010); Kent P. Hymel et al., Mechanisms, Clinical Presentations, Injuries, and Outcomes from Inflicted Versus Noninflicted Head Trauma during Infancy: Results of a Prospective, Multicentered, Comparative Study, 119 Pediatrics 922, 922 (2007); K. Hymel et al., Head Injury Depth as an Indicator of Causes and Mechanisms, 125 Pediatrics 712, 715-18 (2010).

584 Id.

585 See Matthieu Vinchon et al., Accidental and Nonaccidental Head Injuries in Infants: A Prospective Study, 102 J. Neurosurgery: Pediatrics 380, 380-81 (2005); Hymel et al., supra note 581; S. Maguire, Which Clinical Features Distinguish Inflicted from Non-Inflicted Brain Injury? A Systematic Review, 94 Archives Disease Childhood 860, 860 (2009); Gaurav Bhardwaj et al., A Systematic Review of the Diagnostic Accuracy of Ocular Signs in Pediatric Abusive Head Trauma, 117 Ophthalmology 983, 987 (2010); Shruti Agrawal et al., Prevalence of Retinal Hemorrhages in Critically Ill Children, 6 Pediatrics 129, 1388-96 (2012).

586 Joeli Hettler & David S. Greenes, Can the Initial History Predict Whether a Child with a Head Injury has been Abused?, 111 Pediatrics 602, 602 (2003); Duhaime et al., supra note 581; Heather T. Keenan et al., Child Outcomes and Family Characteristics 1 Year After Severe Inflicted or Noninflicted Traumatic Brain Injury, 117 Pediatrics 317, 317 (2006).

587 See Narang, supra note 10, at 541-61.

588 See id.

589 See id.

590 See Findley et al., supra note 3, at 274-75.

591 Id. at 286-90.

592 See supra Part 3.

593 Id.

594 Id.

595 See supra Part 4.

596 See supra Part 5.

597 Id.

598 Id.

599 See supra Part 6; see also Narang, supra note 10, at 563-68. But see Irene Scheimberg et al., Non-Traumatic Intradural and Subdural Hemorrhage and Hypoxic Ischaemic Encephalopathy in Fetuses, Infants and Children Up to 3 Years of Age. Analysis of Two Audits of 636 Cases From Two Referral Centers in the UK, 16 Pediatric & Dev. Pathology 149 (2013).

600 See supra Part 6; Ana Isabel Curcoy et al., Retinal Hemorrhages and Apparent Life-Threatening Events, 26 Pediatric Emergency Care 118 (2010); Raymond D. Pitetti et al., Prevalence of Retinal Hemorrhages and Child Abuse in Children Who Present With an Apparent Life-Threatening Event, 110 Pediatrics 557 (2002); Amy Odom et al., Prevalence of Retinal Hemorrhages in Pediatric Patients After In-Hospital Cardiopulmonary Resuscitation: A Prospective Study, 99 Pediatrics e3 (1997); Charanjit Kaur et al., Early Response of Neurons and Glial Cells to Hypoxia in the Retina, 47 Investigative Ophthalmology & Visual Sci. 1126 (2006); Taiji Nagaoka et al., The Effect of Nitric Oxide on Retinal Blood Flow During Hypoxia in Cats, 43 Investigative Ophthalmology & Visual Sci. 3037 (2002).

601 See supra Part 6; see also Narang, supra note 10, at 505, 588-89; Michael Goldman et al., Severe Cough and Retinal Hemorrhage in Infants and Young Children, 148 J. Pediatrics 835 (2006); Sandra Herr et al., Does Valsalva Retinopathy Occur in Infants? An Initial Investigation in Infants With Vomiting Caused by Pyloric Stenosis, 113 Pediatrics 1658 (2004).

602 Narang, supra note 10, at 574-76.

603 See Findley et al., supra note 3, at 274. The critics have also lodged complaints of "observer bias" and "interpretive errors." However, these relate more to the "minor premise" and, thus, will be discussed in further detail below.

604 See Tuerkheimer, supra note 3, at 13.

605 See Findley et al., supra note 3, at 274.

606 "Circularity" is the logical fallacy in which the manner of proposing a question presumes an answer. For example, assume that scientists wished to prove that a pro sports team can win more games by hiring athletes who have, themselves, won many games. Using a single season's data, they compute the number of times each athlete has won or lost; they then compare these results to the number of times each team has won or lost. The design is clearly invalid--when a team wins, all its players win as well. The predictor variable (number of games the athlete has won) is simply a proxy for the variable being predicted. Consequently, a circular study tends to overstate the strength of an association.

607 See Narang, supra note 10, at 561-62.

608 See Am. Acad. of Pediatrics, Inflicted Childhood Neurotrauma: Proceedings of a Conference Sponsored by Department of Health and Human Services, National Institute of Health, National Institute of Child Health and Human Development, Office of Rare Disease, and National Center for Medical Rehabilitation Research (Robert M. Reece & Carol E. Nicholson eds., 2003).

609 See Duhaime, supra note 581; Hymel, supra note 581.

610 See Vinchon, supra note 442.

611 See Narang, supra note 10, at 523-29.

612 See supra Part 3 A-B; see also M. Shah et al., Motor Vehicle Crash Brain Injury in Infants and Toddlers: A Suitable Model for Inflicted Head Injury?, 29 Child Abuse & Neglect 953, 954 (2005); Feldman et al., supra note 580, at 636; Hobbs et al., supra note

579, at 953; Tzioumi & Oates, supra note 579, at 1105; J. Urban et al., Motor Vehicle Crash-Related Subdural Hematoma From Real-World Head Impact Data, 29 J. Neurotrauma 2774, 2774 (2012).

613 See Feldman et al., supra note 580, at 638; Matschke et al., supra note 580, at 1587.

614 See Narang, supra note 10, at 628.

615 Id. at 596-627.

616 See Findley et al., supra note 3, at 274-80.

617 See Chadwick, supra note 39, at 1213.

618 See generally Kravitz, Williams, Helfer, Lyons, Nimityongskul, Levene, Ruddick, Schaeffer, supra notes 61-72.

619 See Finnie et al., supra note 285, at 237-38.

620 See supra Biomechanics, at 33-45.

621 See Narang, supra note 10, at 551 (citing Amy Odom et al., Prevalence of Retinal Hemorrhages in Pediatric Patients After In-Hospital Cardiopulmonary Resuscitation: A Prospective Study, 99 Pediatrics 4 (June 1997)).

622 See Findley et al., supra note 3, at 296.

623 Stedman's Medical Dictionary (28th ed. 2006).

624 See Wong et al., supra note 541, at 690-91.

625 See Bland v. Verizon Wireless, L.L.C., 538 F.3d 893, 897 (8th Cir. 2008) (stating that "a 'differential diagnosis [is] a technique that identifies the cause of a medical condition by eliminating the likely causes until the most probable cause is isolated.'"); Wilson v. Taser Int'l, Inc. 303 Fed. App'x 708, 714 (11th Cir. 2008) ("[N]onetheless, Dr. Meier did not perform a differential diagnosis or any tests on Wilson to rule out osteoporosis and these corresponding alternative mechanisms of injury. Although a medical expert need not rule out every possible alternative in order to form an opinion on causation, expert opinion testimony is properly excluded as unreliable if the doctor 'engaged in very few standard diagnostic techniques by which doctors normally rule out alternative causes ....").

626 Feit v. Great West Life & Annuity Ins. Co., 271 Fed. App'x 246, 254 (3d Cir. 2008) ("[A]lthough this Court generally recognizes differential diagnosis as a reliable methodology, the differential diagnosis must be properly performed in order to be reliable. To properly perform a differential diagnosis, an expert must perform two steps: (1) 'Rule in' all possible causes of Dr. Feit's death and (2) 'Rule out' causes through a process of elimination whereby the last remaining potential cause is deemed the most likely cause of death.") (citations omitted).

627 See Wong et al., supra note 541, at 691.

628 See Jerome P. Kassirer et al., Learning Clinical Reasoning (2d ed. 2009) ("Bayesian analysis assembles a complete set of diagnostic hypotheses that can explain a given set of clinical findings. For each hypothesis, a set of relevant attributes is identified (historical findings, physical findings, complications, predisposing factors, laboratory results) that might help discriminate among the diagnoses. The prior probability of each diagnostic hypothesis is specified numerically, as is the probability that each attribute is found in each disease entity. Then, a calculation is made of the likelihood of each disease entity given the disease prevalence and the probability of each clinical attribute."). Although physician reasoning does not exclusively proceed in a Bayesian fashion, physicians do frequently rely on Bayesian reasoning (combining disease prevalence with their knowledge of frequency of signs and symptoms in a given disease) in the diagnostic process. See also Wong et al., supra note 541 at 708.

629 See J. Kassirer & F. Sonnenberg, The Scientific Basis of Diagnosis, in Textbook of Internal Medicine (W.N. Kelley ed., 1989); see also A. Elstein & A. Schwartz, Clinical Problem Solving and Diagnostic Decision Making: Selective Review of the Cognitive Literature, 324 Brit. Med. J. 729, 730 (2002).

630 See Wong et al., supra note 541, at 705-06.

631 Id. at 705.

632 Id. at 706. The process of hypothesis refinement is an "evolving, sequential process of data gathering and interpretation." See Kassirer et al., supra note 519, at 5-6.

633 See Kassirer et al., supra note 626, at 5-6. Probabilistic reasoning is Bayesian-type reasoning where prior probabilities of diseases are considered and combined with a physician's knowledge of the frequency of signs and symptoms in a given disease and the probabilities of specific test information. These assist the physician in a probabilistic assessment of the most likely hypothesis. Causal reasoning is a "function of the anatomical, physiological and biochemical mechanisms that operate in normally in the human body and the pathophysiologic behavior of these mechanisms in disease." In assessing causality, physicians use any reliable data, no matter the source. Additionally, temporal proximity can be a potent factor in assessing causation. See J Kassirer & J Cecil, Inconsistency in Evidentiary Standards for Medical Testimony: Disorder in the Courts, 288 J. Am. Med. Ass'n 1382, 1384 (Sep. 18, 2002).

634 "Adequacy" is when the remaining working hypothesis reasonably accounts for all the patient's findings, both normal and abnormal. "Coherency" is when the patient's findings are consistent with the altered pathophysiology of the hypothesized disease. "Parsimony" is the simplest explanation for all of the patient's findings. See Wong et al., supra note 541, at 706-07; see also Kassirer et al., supra note 626, at 5-6.

635 E. Imwinkelried, The Admissibility and Legal Sufficiency of Testimony About Differential Diagnosis (Etiology): Of Under-and Over-Estimations, 56 Baylor L. Rev. 391, 392 (2004).

636 See generally Narang, supra note 10, at 628-29.

637 Id. at 573.

638 See Imwinkelried, supra note 633, at 392.

639 Id.

640 See Best v. Lowe's Home Ctrs. Inc., 563 F.3d 171, 178, 183-84 (6th Cir. 2009) (stating a differential diagnosis can be adequate grounds for a causation opinion under Daubert); Hyman & Armstrong, P.S.C. v. Gunderson, 279 S.W.3d 93, 107 (Ky. 2008); Westberry v. Gislaved Gummi AB, 178 F.3d 257, 263 (4th Cir. 1999).

641 See Gunderson, 279 S.W.3d at 107 (citing Globetti v. Sandoz Pharms. Corp., 111 F.Supp.2d 1174, 1177 (N.D. Ala. 2000)).

642 See State v. McMullen, 900 A.2d at 118 (holding that testimony of two state medical experts regarding Pediatric Condition Falsification was sufficiently relevant and reliable under Daubert when those experts "soundly performed" their differential diagnosis); State v. Edwards, 2011 WL 1378927 at *3 (Ohio Ct. App. April 13, 2011) (holding that the trial court did not abuse its discretion when it concluded expert testimony on AHT/SBS was reliable under Daubert; the court stated that "differential diagnosis is a standard scientific method for determining causation"); State v. Carr, 2010 WL 2473337 at *6 (Ohio Ct. App. June 18, 2010) (stating that "[t]he process of isolating the cause of a patient's injuries through the methodical elimination of other potential causes, called differential diagnosis, is a standard scientific method for determining causation," and expert testimony based upon such was reliable under Daubert); Overton v. State, 2009 WL 3489844 at *46 (Tex. App.--Corpus Christi October 29, 2009) (holding that trial court did not abuse its discretion in finding expert testimony opining that a child died of "non-accidental hypernatremia" reliable under Daubert when that expert based his opinion on "the widely accepted practice of differential diagnosis").

643 See National Research Council, supra note 412, at 4. Although clinical medicine is not considered a classic forensic discipline (such as fingerprint identification, forensic pathology, or bite-mark identification), there are aspects of clinical medicine, such as child abuse pediatrics, that have direct forensic applications. Thus, the concerns raised by the NRC are, at least, tangently relevant to the methodologies employed by child abuse pediatricians.

644 See Imwinkelried, supra note 633, at 392.

645 Id. at 393.

646 Id.

647 See Kassirer et al., supra note 626.

648 See Imwinkelried, supra note 633, at 420 (citing Clausen v. M/V New Carissa, 156 F. Supp. 2d 1192, 1194-95 (D. Or. 2001) (utilizing expert description of process-of-elimination reasoning to determine whether an oil spill caused the death of oysters in a commercial farm)).

649 Id. (citing Karl Popper, The Logic of Scientific Discovery 22 (1959)).

650 See Findley et al., supra note 3, at 286-90.

651 See National Research Council, supra note 544, at 4-9.

652 Id. at 185.

653 Id. at 8-9 (citing P.C. Giannelli, Wrongful Convictions and Forensic Science: The Need to Regulate Crime Labs, 86 N.C. L. Rev. 163, 220-22 (2007)).

654 Id. at 6, 8, 19-20.

655 Id. at 119-20; see also Narang, supra note 10, at 538.

656 See National Research Council, supra note 544, at 120.

657 See Narang, supra note 10, at 538 ("'Sensitivity'[, or the "true positive rate" (TPR) of a particular test,] is 'the probability that a test for a disease will give a positive result' when the patient actually has the disease. Put simply, it is actually the chance the condition will be found by the test.").

658 Id. ("'Specificity' [or the "true negative rate" (TNR) of a particular test,] is 'the probability that a test for disease will give a negative result when the patient does not have the disease.' Put simply, it is the chance that someone without the disease will actually have a negative test.").

659 See Narang, supra note 10, at 538-58.

660 Daniel Kahneman, Lecture at the Princeton University Dep't of Psychology: Maps of Bounded Rationality: A Perspective on Intuitive Judgment and Choice, 449 (Dec. 8, 2002), available at http://nobelprize.org/nobel_ prizes/economics/laureates/2002/kahneman-lecture.pdf.

661 Id. at 450-51.

662 Id. at 450.

663 Id.

664 Id. at 452, 455.

665 See Kassirer, supra notes 626 and 627.

666 See Kahneman, supra note 658, at 455.

667 Id. at 465; see also Arthur Elstein & Alan Schwartz, Clinical Problem Solving and Diagnostic Decision Making: Selective Review of the Cognitive Literature, 324 Brit. Med. J. 729 (2002).

668 Kahneman, supra note 658, at 465.

669 Id. (citing Amos Tversky & Daniel Kahneman, Judgment under Uncertainty: Heuristics and Biases, 185 Sci. 1124-31 (1974)).

670 Kahneman, supra note 658, at 450.

671 Id. at 473.

672 Id. at 455-56 (emphasis added).

673 Id. at 473 (citations omitted).

674 Id. at 465.

675 See Elstein & Schwartz, supra note 665, at 731.

676 See Narang, supra note 10, at 579.

677 Kahneman, supra note 658, at 482.

678 See Elstein & Schwartz, supra note 665, at 731-32.

679 Id. at 731.

680 Id. at 729-31.

681 Narang, supra note 10, at 571-74.

682 Findley et al., supra note 3, at 281.

683 Am. Psychiatric Ass'n, Diagnostic and Statistical Manual of Mental Disorders 589 (4th ed., text revision 2000).

684 Id. at 590.

685 Id. at 592.

686 Id. at 593-94.

687 "Individuals whose binge-eating behavior occurs only during Anorexia Nervosa are given the diagnosis Anorexia Nervosa, Binge-Eating/Purging Type, and should not be given the additional diagnosis of Bulimia Nervosa." Id. at 593 (emphasis in original). However, in certain neurological or other general medical conditions, if the full criteria for Bulimia Nervosa is also met, both diagnoses can be given. Id.

688 Many critics lump AHT/SBS cases into "triad" cases (SDH, RH, and cerebral edema). However, this is over-simplistic and inaccurate. While, certainly, a small percentage of AHT/SBS cases contain only these findings, in many other cases there are other important findings that physicians must account for in the unifying diagnosis.

689 It is important to note at this point that, for the purposes of simplicity in this example, the determination of the medical findings is not disputed, as can be in real-life circumstances. For example, whether a radiographic finding represents a true fracture or is a normal variant of the human body or some other explanation is another consideration in medical decision making that must be made and involves training, experience, and ongoing literature review.

690 Narang, supra note 10, at 628-29.

691 See Reece, supra note 609, at 148-49.

692 Id. at 103.

693 See Kassirer, supra notes 626-27.

694 See R. Behrman, Nelson's Textbook of Pediatrics (17th ed. 2004).

695 See supra Part 4.D.

696 See supra Table 1.

697 Id.

698 Christopher J. Hobbs et al., Subdural Haematoma and Effusion in Infancy: An Epidemiological Study, 90 Arch. Dis. Child 952, 952 (2005); Heather T. Keenan et al., A Population-Based Study of Inflicted Traumatic Brain Injury in Young Children, 290 J. Am. Med. Ass'n 621, 621 (2003); Katherine D. Ellingson et al., Using Hospital Discharge Data to Track Inflicted Traumatic Brain Injury, 34 Am. J. Prev. Med. S157, S157 (2008).

699 See supra Part X.

700 Sabine A. Maguire et al., Retinal Haemorrhages and Related Findings in Abusive and Non-Abusive Head Trauma: A Systematic Review, 27 Eye 28 (2013).

701 Id. at 28-30.

702 Id. at 29.

703 Id.

704 Id. at 29-30.

705 Id. at 31.

706 Id.

707 Id.

708 Sabine A. Maguire et al., Which Clinical Features Distinguish Inflicted from Non-inflicted Brain Injury. A Systematic Review, 94 Arch. Dis. Child 860, 860 (2009).

709 J. Barth et al., Psychosocial Interventions for Smoking Cessation in Patients with Coronary Heart Disease, Cochrane Database of Systematic Reviews 2008, Issue 1. Art. No.: CD006886.

710 Id.

711 Id.

712 See id.

713 See Elstein & Schwartz, supra note 665, at 731-32.

714 The standard for achieving diagnostic sufficiency is undefined. Whether it is a preponderance standard, clear and convincing standard, or beyond a reasonable doubt standard has never been carefully explored or clearly enunciated, in the medical or legal literature. In most circumstances, given clinical exigency and the primacy of treating the patient, the standard most likely approximates a preponderance standard. But this is a topic for further discussion at a different time. What is clear is that Findley et al., and others, have confused it for the standard for legal sufficiency for conviction--beyond a reasonable doubt.

715 See Findley et al., supra note 3, at 215.

716 Id. at 292.

717 Id.

718 Id. at 216, 266, 292, 300.

719 Id. at 301.

720 Id. at 236-37.

721 Id. at 307.

722 Id. at 301.

723 Id.

724 Id. at 305.

725 Id. at 309-12.

726 While Findley et al. also recommend "research" as a path forward (see Findley et al., supra note 3, at 307-08), we agree with some of their delineated research objectives, but disagree with others (such as the establishment of a national registry of AHT/SBS cases).

727 See National Research Council Report, supra note 544.

728 Email communications on file with Dr. Narang.

729 See Hymel et al., supra note 146.

730 See Narang, supra note 10, at 594.

731 See Findley et al., supra note 3, at 309.

732 See Austin v. Am. Ass'n of Neurological Surgeons, 253 F.3d 967, 972-73 (7th Cir. 2001).

733 See Melendez-Diaz v. Mass., 557 U.S. 305, 305-06 (2009).

734 See Joelle Moreno, C.S.I. BULLS#!T: The National Academy of Sciences, Melendez-Diaz v. Massachusetts, and Future Challenges to Forensic Science and Forensic Experts, 2 Utah L. Rev. 327, 330 (2010).

735 See Hand, supra note 516, at 56 (calling for "a board of experts or a single expert, not called by either side," to "advise the jury of the general propositions applicable to the case").

736 See Findley et al., supra note 3, at 306.

737 See id. at 213.

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